

Award Number: W81XWH-08-1-0544

TITLE: 26th National Neurotrauma Symposium

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REPORT DATE: December 2008

TYPE OF REPORT: Final Proceedings

PREPARED FOR: U.S. Army Medical Research and Materiel Command
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1. REPORT DATE (DD-MM-YYYY) 12/31/2008		2. REPORT TYPE Final Proceedings		3. DATES COVERED (From - To) 25 Jul 2008 - 30 Jul 2008	
4. TITLE AND SUBTITLE 26th National Neurotrauma Symposium				5a. CONTRACT NUMBER	
				5b. GRANT NUMBER W81XWH-08-1-0544	
				5c. PROGRAM ELEMENT NUMBER	
6. AUTHOR(S) Edward D. Hall, Ph.D., Email: edhall@uky.edu				5d. PROJECT NUMBER	
				5e. TASK NUMBER	
				5f. WORK UNIT NUMBER	
7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES) National Neurotrauma Society Attn: Linda Garcia, Executive Director 8032 SW 45 Lane Gainesville FL 32608 USA				8. PERFORMING ORGANIZATION REPORT NUMBER	
9. SPONSORING / MONITORING AGENCY NAME(S) AND ADDRESS(ES) USAMRMC				10. SPONSOR/MONITOR'S ACRONYM(S) USAMRMC	
				11. SPONSOR/MONITOR'S REPORT NUMBER(S)	
12. DISTRIBUTION / AVAILABILITY STATEMENT Approved for public release; distribution unlimited					
13. SUPPLEMENTARY NOTES					
14. ABSTRACT This report concerns the 26 th Annual National Neurotrauma Symposium which was held from July 25-30, 2008 at the Hilton-Disneyworld Resort in Lake Buena Vista, Florida for which a \$10,000 grant was received from the U.S. Army Medical Research and Materials Command. The symposium which, was jointly sponsored by the National Neurotrauma Society and the Section on Neurotrauma & Critical Care of the American Association of Neurological Surgeons/Congress of Neurological Surgeons, attracted 609 registrants which included a mix of neurotrauma-focused basic scientists and clinicians (neurologists, neurosurgeons, neurointensivists, psychiatrists) from both academic, industrial and military institutions. Plenary sessions covered the following topics: Clinical Update on Management of Traumatic Brain Injury (TBI), Spinal Cord Injury (SCI) Regeneration, Pediatric TBI, Blast TBI, Nutrition in SCI and TBI, Oxidative Damage in SCI and TBI, Glial Cell Responses and Interactions, ICU Management and Monitoring, Mild TBI and NIH Initiatives in TBI Research. In addition, there were four sessions devoted to 15 min. open communications and 4 poster sessions which included 326 posters. The abstracts were published in the Journal of Neurotrauma.					
15. SUBJECT TERMS Neurotrauma, Spinal Cord Injury, Traumatic Brain Injury					
16. SECURITY CLASSIFICATION OF: U			17. LIMITATION OF ABSTRACT UU	18. NUMBER OF PAGES 81	19a. NAME OF RESPONSIBLE PERSON USAMRMC
a. REPORT U	b. ABSTRACT U	c. THIS PAGE U			19b. TELEPHONE NUMBER (include area code)

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INTRODUCTION

The National Neurotrauma Symposium is an annual meeting of neuroscientists, clinicians, research fellows and graduate and medical students who are involved in research and treatment of patients suffering from the effects of central nervous system (CNS) trauma. The National Neurotrauma Symposium is the premier annual meeting of the Neurotrauma community, as attested to by the choice of the Symposium for the presentation of the prestigious Ameritec Prize, given to scientists whose research contributes significantly to the cure of paralysis. The National Neurotrauma Symposium provides the opportunity for basic scientists and clinicians and allied health workers to meet and discuss questions related to the pathophysiology of brain and spinal cord injury, as well as mechanisms of recovery following trauma. The National Neurotrauma Symposium is unique in both the subject matter that is discussed and the diversity of the attendees. The National Neurotrauma Symposium is organized under the auspices of the National Neurotrauma Society, the preeminent professional organization for scientists and clinicians involved in Neurotrauma research. As in previous meetings, the 2008 National Neurotrauma Symposium focused specifically on areas of debate within the research communities of central nervous system injury and repair primarily related to traumatic brain and spinal cord injury.

For the 2008 symposium, the NNS partnered with the Section on Neurotrauma & Critical Care of the American Association of Neurological Surgeons and Congress of Neurological Surgeons (AANS/CNS). As a result, four clinical breakout sessions were added to the usual meeting format. These did not replace the more basic research-focused programmatic themes, but rather the clinical sessions were held in parallel with certain basic science sessions. This collaboration with the AANS/CNS increased the attendance and participation of clinical neurotraumatologists, enhanced the translational value of the symposium and provided an opportunity for civilian and military basic and clinical neurotraumatologists to interact with great benefit to both.

The **Specific Aims** of the 26th annual National Neurotrauma Symposium were:

1. To provide a forum for the presentation, discussion and feedback regarding the most recent findings in Neurotrauma research and to encourage interaction between junior and senior investigators in the field.
2. To encourage the participation and education of students studying in the field of Neurotrauma. The Symposium fosters student participation through large poster sessions, student oral open-communication sessions, student poster competition, social mixers, student travel awards, and subsidized student registration fees.
3. To make the meeting more relevant to clinical neurotrauma researchers, to increase the real world neurotrauma knowledge base for basic neurotrauma scientists and thereby enhance basic-clinical interactions and translationally-focused two way collaboration ("Bench to Bedside" and "Bedside to Bench").
4. To continue to encourage the involvement and advancement of women, minority groups, and persons with disabilities in Neurotrauma research. The Symposium is the forum for scientific and social activities hosted by Women in Neurotrauma Research, the professional society for women in clinical and laboratory Neurotrauma research.
5. To have relevant and thought-provoking presentations by researchers not directly involved in Neurotrauma research. The goal is to foster new ideas and ways of thinking into mainstream Neurotrauma as well as to encourage scientist from other disciplines to engage in Neurotrauma research.

The entire symposium had a high degree of relevance to the USAMRMC Mission considering the fact that acute neurotrauma is a major unmet medical need for our warfighters in Iraq, Afghanistan and elsewhere. Each of the sessions, basic and clinical, showcased cutting edge science that is translatable to the battlefield with the aim of decreasing mortality and morbidity in military personnel. The scientific program included a presentation by Col. Geoffrey Ling in the Blast Injury session, a presentation by Captain James Ecklund in the Surgical Decompression for TBI session and one by Colonel Charles Hoge in the session on Mild TBI. In addition, Dr. Frank Tortella (Walter Reed) will chaired the Blast Injury session and served as a member of the Program Committee.

BODY

This report concerns the 26th Annual National Neurotrauma Symposium which was held from July 25-30, 2008 at the Hilton-Disneyworld Resort in Lake Buena Vista, Florida for which a \$10,000 grant was received from the U.S. Army Medical Research and Materials Command. The symposium which, was jointly sponsored by the National Neurotrauma Society and the Section on Neurotrauma & Critical Care of the American Association of Neurological Surgeons/Congress of Neurological Surgeons (AANS/CNS), attracted 609 registrants which included a mix of neurotrauma-focused basic scientists and clinicians (neurologists, neurosurgeons, neurointensivists, psychiatrists) from both academic, industrial and military institutions. Plenary sessions covered the following topics: Clinical Update on Management of Traumatic Brain Injury (TBI), Spinal Cord Injury (SCI) Regeneration, Pediatric TBI, Blast TBI, Nutrition in SCI and TBI, Oxidative Damage in SCI and TBI, Glial Cell Responses and Interactions, ICU Management and Monitoring, Mild TBI and NIH Initiatives in TBI Research. In addition, there were four sessions devoted to 15 min open communications and 4 poster sessions which included 326 posters. The abstracts were published in the Journal of Neurotrauma. There was also a student

poster competition that included the top 16 poster abstracts. The final program is provided in **APPENDIX I**. The results of the symposium evaluation which were uniformly positive are presented in **APPENDIX II**.

KEY RESEARCH ACCOMPLISHMENTS

- State of the art reviews of the latest basic and clinical science and clinical management of TBI and SCI in 10 plenary sessions for 609 basic scientist and clinician participants from academic, industrial and military attendees
- Presentation of 326 posters on various neurotrauma topics
- Student poster competition and awards honoring top 16 student/postdoctoral fellow poster abstracts and presentations.

REPORTABLE OUTCOMES

- Plenary speaker, open communication speaker and poster abstracts from the symposium were published in the Journal of Neurotrauma 25, Number 7, 2008 and available for online access at website of Marianne Liebert Publishers, Inc. (www.liebertonline.com).

CONCLUSION

With the help of the much appreciated \$10,000 in financial support from the USAMRMC, the 26th Annual National Neurotrauma Symposium was a major success and provided a clear benefit to all of the scientific attendees. The enhanced participation of neurotrauma clinicians and clinician scientists greatly increased the translational value of the symposium and the much needed interaction between basic scientists and clinicians working in the neurotrauma field. This has resulted in an ongoing relationship and future joint meeting sponsorship by the National Neurotrauma Society and the AANS/CNS Section on Neurotrauma & Critical Care. In that regard, the next meeting will be held in Santa Barbara, CA and is being organized by Dr. David Hovda, current President of the National Neurotrauma Society and Director of the UCLA Brain Injury Research Center. It will be jointly sponsored by the National Neurotrauma Society, the AANS/CNS Section on Neurotrauma & Critical Care and the International Neurotrauma Society and will be held from September 7-11, 2009. The 26th National Neurotrauma Symposium and future symposiums serve to increase communication between basic and clinical neurotraumatologists and to speed the design and delivery of improved acute and chronic care for civilian and military personnel who sustain central nervous system injuries.

REFERENCES

Journal of Neurotrauma 25(7):853-935, 2008

APPENDICES

- I. Final program for 26th National Neurotrauma Symposium
- II. Evaluation Results for 26th National Neurotrauma Symposium

Appendix I- Final Program for 26th National Neurotrauma Symposium

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LETTER FROM THE NNS PRESIDENT

July 2008

Dear Neurotrauma Colleagues:

Greetings and welcome to the 2008 National Neurotrauma Symposium, the 26th annual scientific meeting sponsored by the National Neurotrauma Society (NNS). For the 2008 symposium, the NNS is partnering with the American Association of Neurological Surgeons and Congress of Neurological Surgeons (AANS/CNS) Section on Neurotrauma and Critical Care. This collaboration with the AANS/CNS has been directed at increased participation and interaction with clinical neurotraumatologists, enhanced translational and collaborative value of the symposium, providing a better opportunity for basic and clinical neurotraumatologists to exchange science with great benefit to both groups.



The goals of the 26th Annual National Neurotrauma Symposium are:

6. To provide a forum for the presentation, discussion and feedback regarding the most recent findings in Neurotrauma research and to encourage interaction between junior and senior investigators in the field.
7. To encourage the participation and education of students studying in the field of Neurotrauma. The Symposium fosters student participation through large poster sessions, student oral open-communication sessions, student poster competition, social mixers, student travel awards, and subsidized student registration fees.
8. To make the meeting more relevant to clinical neurotrauma researchers, to increase the real world neurotrauma knowledge base for basic neurotrauma scientists and thereby enhance basic-clinical interactions and translationally-focused two way collaboration ("Bench to Bedside" and "Bedside to Bench").
9. To continue to encourage the involvement and advancement of women, minority groups, and persons with disabilities in Neurotrauma research. The Symposium is the forum for scientific and social activities hosted by Women in Neurotrauma Research, the professional society for women in clinical and laboratory Neurotrauma research.
10. To have relevant and thought-provoking presentations by researchers not directly involved in Neurotrauma research. The goal is to foster new ideas and ways of thinking into mainstream Neurotrauma as well as to encourage scientist from other disciplines to engage in Neurotrauma research.

The Program Committee, listed in the program guide, has constructed an excellent scientific agenda that includes 10 basic science-focused plenary sessions. Among these are sessions devoted to important and timely neurotrauma topics that have not been covered at previous meetings including "*Blast TBI in Combat Casualty Care and Civilian Terrorism*", "*Nutrition in Spinal Cord & Brain Injury*" and "*Mild Traumatic Brain Injury*". In addition, other sessions are focused on topics that have not been included in the meeting for several years such as "*Newer Perspectives on the Role of Oxidative Damage in TBI and SCI*". As a result of our collaboration with the AANS/CNS Section on Neurotrauma and Critical Care, 4 clinically focused sessions, three of which will be held as breakouts in parallel with basic science sessions have been added to the program. The clinical topics are "*Clinical Update on Management of TBI*", "*Clinical Update on Management of SCI*", "*Surgical Decompression in TBI*" and "*ICU Management and Advanced Neuromonitoring*". In regards to both the basic and clinical sessions, the Program Committee has tried to give equal time to the TBI and SCI subfields.

In addition to the plenary and clinical breakout sessions, 4 poster sessions are included and structured as in last year's meeting. As part of this, the annual student poster competition and awards will continue to be a highlight of the meeting. There are also 4 open communication sessions (2 TBI, 2 SCI) consisting of 4 speakers each. These have been selected from the submitted abstracts based upon their having been judged to be of the highest scientific quality and interest. I am extremely grateful to NNS Vice President Kathryn (Kathy) Saatman who has worked hard to organize the poster competition and abstract selection for the open communication sessions with the help of a host of volunteer faculty reviewers. In addition, I most enthusiastically thank our meeting organizer Karen Gottlieb and her colleagues at TLC Events Group, Inc. for their incredibly competent, thorough, friendly and dedicated work in organizing the symposium. Last, but certainly not least, I am extremely appreciative of the dedication and generous efforts of our NNS Executive

Director Linda Garcia in her handling of NNS business and her assistance with the meeting organization and communication with the NNS membership. Please remember to thank Kathy, Karen and Linda when you see them during the meeting.

In addition to their annual luncheon meeting, the Women In Neurotrauma Research (WINTR), under the leadership of Dr. Christina Marmarou, will again host a session on Tuesday evening that will provide an opportunity for young scientists to be paired up with more senior and well-established investigators.

Financial support from federal and foundation grants, institutions, exhibitors and sponsors which are highlighted in this Program Guide enable the NNS to present the symposium year after year and to provide student travel awards. The meeting would be impossible to host without this continued support.

While we are all here to communicate and advance the latest research findings in basic and clinical neurotrauma research, we have chosen the present meeting venue so that participants and their friends and families could also have a most enjoyable break from their hard work. Accordingly, please take time to enjoy yourselves here at the Hilton Walt Disney World Resort and the Disney World theme parks. Even your President is going to fit in as much Disney World as he finds time for.

Once again, thank you for being a part of the success of the 2008 National Neurotrauma Symposium. I am certain you will find it to be an exciting and highly intellectually stimulating meeting in a totally fun and enjoyable location.

Lastly, I hope you will all participate in next year's symposium which will be a joint meeting of the National and International Neurotrauma Societies, again in conjunction with the AANS/ CNS Section on Neurotrauma and Critical Care to be held in Santa Barbara, California from September 7-11, 2009 that is being organized by NNS President-Elect David Hovda who will provide details at the end of this year's symposium.

With my warmest regards,



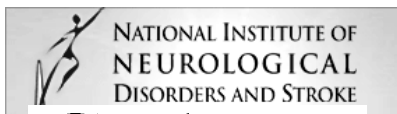
Edward D. Hall
President, National Neurotrauma Society

SUPPORTERS & EXHIBITORS



The National Neurotrauma Society and the School of Medicine at Virginia Commonwealth University gratefully acknowledge the following supporters for their generous contributions:

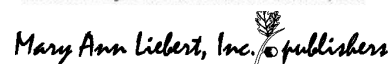
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These presenters report having the following relevant relationships to disclose:

David P. Adelson is an investigator for NIH and on the Scientific Advisory Board for TraumaTec, Inc.

Michael Fehlings is a consultant for DePuy Spine and Alsius Pharmaceuticals.

David O. Okonkwo has received honoraria from Alsius, Medtronic, and Synthes.

Patrick Kochanek is a patent holder for an emergency preservation & resuscitation product.

Brian Kwon is a consultant for Medtronic.

Geoff Manley receives grant funding from the NIH.

Andrew Maas receives grand funding from the NIH/NINDS.

These presenters report having no relevant relationships to disclose:

Bizhan Aarabi
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Robert S. B. Clark
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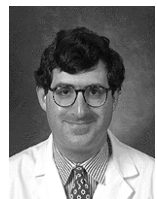
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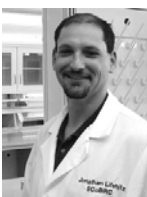
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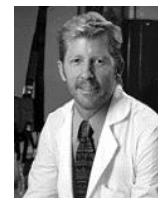
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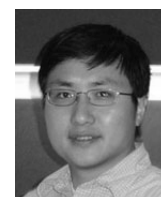
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All abstract posters will be displayed in the International Ballroom.

Please be sure you are available at your poster during the scheduled session time to present your abstract. Although you are not *required* to remain at your poster board location unless you are a Top 16 finalist (Sessions A&B only), you are welcome to present your poster at both assigned sessions.

Be sure to set up and remove your posters during the times indicated below.

Posters left remaining after the session removal period will be discarded – NNS takes no responsibility for posters.

Poster Numbers	Poster Session	Session Time	Set up	Removal
13, 86, 138, 147, 153, 176, 226, 232	Top 16 Student Abstracts Group A	JUDGING SESSION A Monday 9:45 AM - 11:00 AM	After 6:00 PM on Sunday All Top Student Abstracts will be on display for the entire meeting.	Wednesday by 12:00 PM
21, 96, 104, 190, 191, 206, 272, 290	Top 16 Student Abstracts Group B	JUDGING SESSION B Monday 2:30 – 3:45 PM		
1-82	Session A Monday	9:45 AM - 11:00 AM	After 6:00 PM on Sunday Or Monday before 9:45 AM	Monday between 11:00 AM – 1:00 PM
83-164	Session B Monday	2:30 – 3:45 PM	Between 11:00 AM and 2:30 PM on Monday	Monday between 5:15 - 6:45 PM
165-245	Session C Tuesday	9:30 – 10:45 AM	After 5:15 PM Monday Or Tuesday before 9:30 AM	Tuesday between 10:45 AM – 1:00 PM
246-326	Session D Tuesday	2:15 – 3:30 PM	Between 10:45 AM and 2:15 PM on Tuesday	Tuesday after 3:30 PM

ORAL PRESENTATIONS

Each abstract selected for oral presentation will be allotted 10 minutes for their presentation, with an additional 5 minutes for Q&A, for a total of 15 minutes. Please be sure to arrive at your designated room at least 15 minutes prior to your presentation session with your presentation on a PC formatted disk in order to coordinate with the A/V technician. Please refer to the Schedule of Events for the date and time of your presentation.

TRAVEL GRANTS

Thanks to generous support in the form of a grant from the National Institutes of Health (NIH), the National Neurotrauma Society is able to offer \$10,000 in travel grants to encourage students to attend the conference. The travel grants are awarded based on financial need and merit.

We are pleased to announce that this year's NNS Travel Grant awardees are:

Pamela Harvey
Melissa Laird
Laura Gonzalez-Lara
Jordan Clark
Teddy Youn

Maxine Reger
Melissa Simon
Sakina G. Thawer
Kyle Fink
Lai Yee Leung

Wei Jin
Qin Chen
Anders Hanell
Eugene Park
Cassie Mitchell

Yong Jiang
Alexander Tuchman
Laura Sundberg
Tracy Yuen
Zin Khaing

Thanks also to generous support in the form of a grant from Synthes CMF, we are also able to award an additional seven travel grants specifically for neurosurgical residents.

The Synthes Travel Grant awardees for 2008 are:

Hong Yan
Joshua Anderson

Pawel Ochalski
Rodney Samuelson

Sorin Craciunas
Gregory Hawryluk

Sandya Venugopal

GENERAL INFORMATION

REGISTRATION & INFORMATION DESK

The Registration Desk will be open from 12:00 PM to 6:00 PM Sunday, 7:00 AM to 5:00 PM Monday and Tuesday, and 7:00 AM to 1:00 PM on Wednesday. Conference staff will be on hand to assist participants in every possible way, so as to ensure your NNS experience is both enjoyable and rewarding.

Proof of Attendance certificates (*non-CME*) will automatically be emailed to all attendees after the conference.

FUTURE MEETINGS

The NNS Conference website at www.neurotrauma.org/2008 will be updated with Conference highlights, awards and winners of the Poster Competition following the completion of this years' conference.

Next years' symposium will be a joint meeting held in conjunction with the International Neurotrauma Society and the AANS/CNS Section on Neurotrauma & Critical Care. This 4-day meeting will be held next September 7-11, 2009 at Fess Parker's Doubletree Resort in Santa Barbara, California. Complete details for **Neurotrauma 2009: The Second Joint Symposium of the National & International Neurotrauma Societies** including the AANS/CNS Section on Neurotrauma & Critical Care will be posted online soon at www.neurotrauma.org/2009.

NAME BADGES

PLEASE WEAR YOUR NAME BADGE AT ALL TIMES.

Name badges are required for access to all sessions, meals and evening events included with registration.

Be sure to bring your ticket with you for admittance to the WINTR Reception.

CME CREDIT DESIGNATION

This activity has been planned and implemented in accordance with the Essential Areas and policies of the Accreditation Council for Continuing Medical Education (ACCME) through the joint sponsorship of the Virginia Commonwealth University School of Medicine (VCU) and the National Neurotrauma Society. VCU is accredited by the ACCME to provide continuing medical education for physicians.

Physicians may claim up to 18.5 hours in Type 1 or Type 2 CME on the Virginia Board of Medicine Continued Competency and Assessment Form required for renewal of an active medical license.

VCU designates this educational activity for a maximum of **18.5 AMA PRA Category 1 Credits™**. Physicians should only claim credit commensurate with the extent of their participation in the activity.

RESPONSIBILITY

By registering and participating in the NNS Symposium, the attendee shall hold harmless the National Neurotrauma Society, National Neurotrauma Symposium, Inc., NNS Administration and elected officials, Program Chairs and Conference Organizers in the case of any damage or personal injury claims. In addition, none of the parties shall be liable for non-performance including but not limited to, strikes or labor unrest, delay in transportation, delay in delivery by suppliers, fire, wars, acts of governments, unavailability of power or other utilities, or acts of nature.

GENERAL INFORMATION

HILTON IN THE WALT DISNEY WORLD RESORT

1751 Hotel Plaza Boulevard, Lake Buena Vista, Florida, United States 32830
Tel: 1-407-827-4000 Fax: 1-407-827-3890

Check-In: 3:00 PM - Check-Out: 11:00 AM

The Hilton, located in the Walt Disney World® Resort, is an official hotel of Walt Disney World in Lake Buena Vista, Florida. Hotel guests will enjoy exceptional facilities at AAA's longest-running Four Diamond Resort in Central Florida. Our official Walt Disney World hotel is only steps away from some of Walt Disney World's most popular amenities and attractions, including: Disney's Magic Kingdom®, Disney's Animal Kingdom® Theme Park, Disney's Hollywood Studios™, Disney's Epcot®, Disney's Typhoon Lagoon® Water Park, Disney's Blizzard Beach® Water Park, Downtown Disney® Marketplace, Downtown Disney® Pleasure Island, and Downtown Disney® Westside.

AIRPORT SHUTTLE RESERVATIONS

Orlando Airport and Attraction Shuttle Reservations
Mears Transportation Group
(407) 423-5566

WOMEN IN NEUROTRAUMA EVENTS

WINTR BrainSTORM Business Meeting

Monday, July 28th
5:45 PM - 6:45 PM

*All WINTR members are
invited to attend!*

WINTR Luncheon Special Session:

"Translational Research Grants in Neurotrauma"

Dr. Ramona Hicks, Ph.D.
NIH/NINDS Program Director
Tuesday, July 29th
11:45 AM - 12:45 PM

**Boxed lunches will be
available for purchase*

WINTR Reception & Mentoring Program**

Tuesday, July 29, 2008
5:00 PM- 7:00 PM

*"Speed Mentoring" Reception -
Laughter and discussion between
protégé and mentor. Learn from
leading researchers across the field or
lend advice to aspiring researchers!*

***Advanced Ticket purchase required*

LUNCH OPTIONS – JULY 29th

Lunch items will be available for purchase in the Grand Foyer prior to the WINTR Special Session and may be taken into the meeting. Sample "Sidewalk Café" items will include: Fruit Salad (\$5), Caesar Salad (\$5), Assorted Sandwiches (\$6.50), Cookies (\$2), Dry Snacks (\$2) and drinks.

EVENING SOCIAL EVENTS

STUDENT NETWORKING HAPPY HOUR

Sunday, July 27th
Rum Largo Pool Bar
5:30 – 7:00 pm

**Drinks & Snacks may be charged
directly to your room or credit card*

WELCOME RECEPTION

Monday, July 28th
Grand Ballroom Foyer
6:45 – 8:15 PM

*All delegates & registered
accompanying spouses
are invited to attend*

WINTR RECEPTION & MENTORING PROGRAM

Tuesday, July 29th
Palm Ballroom 3
5:00 – 7:00 PM

(Advanced purchase tickets only)

ON PROPERTY DINING OPTIONS

At the Hilton WDW Resort:

- **Andiamo Italian Bistro & Grill** - Bistro and wine bar offering an authentic and unique dining experience. Freshly prepared house specialties including hot and cold antipasti, homemade ravioli, incredible pasta selections and traditionally prepared meat and grilled fish dishes compliment award-winning service. Featuring an extensive wine list and full bar, and a children friendly menu. For reservations, please call (407) 827-3838. Open 5:30 pm – 11:00 pm daily.
- **Covington Mill** - Casual dining restaurant. Enjoy a sumptuous Breakfast Buffet and Lunch Specials daily including a variety of salads, pastas, sandwiches, and signature dishes in a casual setting. On Sundays, take your kids to see the Disney Character Breakfast, hosted from 8:30am to 11:00am.
- **Benihana Steakhouse and Sushi** - Tempt your taste buds and be entertained at Benihana! Our chefs will perform before you as they cook according to a 1,000-year-old Japanese recipe. Menu selections ranging from chicken and steak to sushi and sashimi. Open 4:00 pm-10:00 pm Monday - Thursday, 2:00 pm-10:00 pm Friday-Sunday.
- **John T's Lounge** - Cheer a favorite sports team while enjoying beer from around the world. Light snacks are available for lunch and dinner. Open 3:00 pm – 2:00 am daily.
- **Rum Largo Poolside Bar & Cafe** - Experience the tropics at Rum Largo Poolside Bar & Café. Take a break from sunbathing with an ice-cold, frozen drink. Groove to the rhythms of Key West-style music. Our full-service bar and café also offers a menu of delectable salads, sandwiches, and pizzas. Open 11:00 am – 6:00 pm daily.
- **Mugs** - Offering specialty coffees, gelato, ice cream, wine and beer. Open 4:00 pm – midnight daily.
- **Mainstreet Market** - Mainstreet Market features a gourmet deli, Starbucks coffee, salads, sandwiches, snacks, fresh fruits and ice cream. Open 24 hours.
- **24 hour In-Room Dining is also available.**

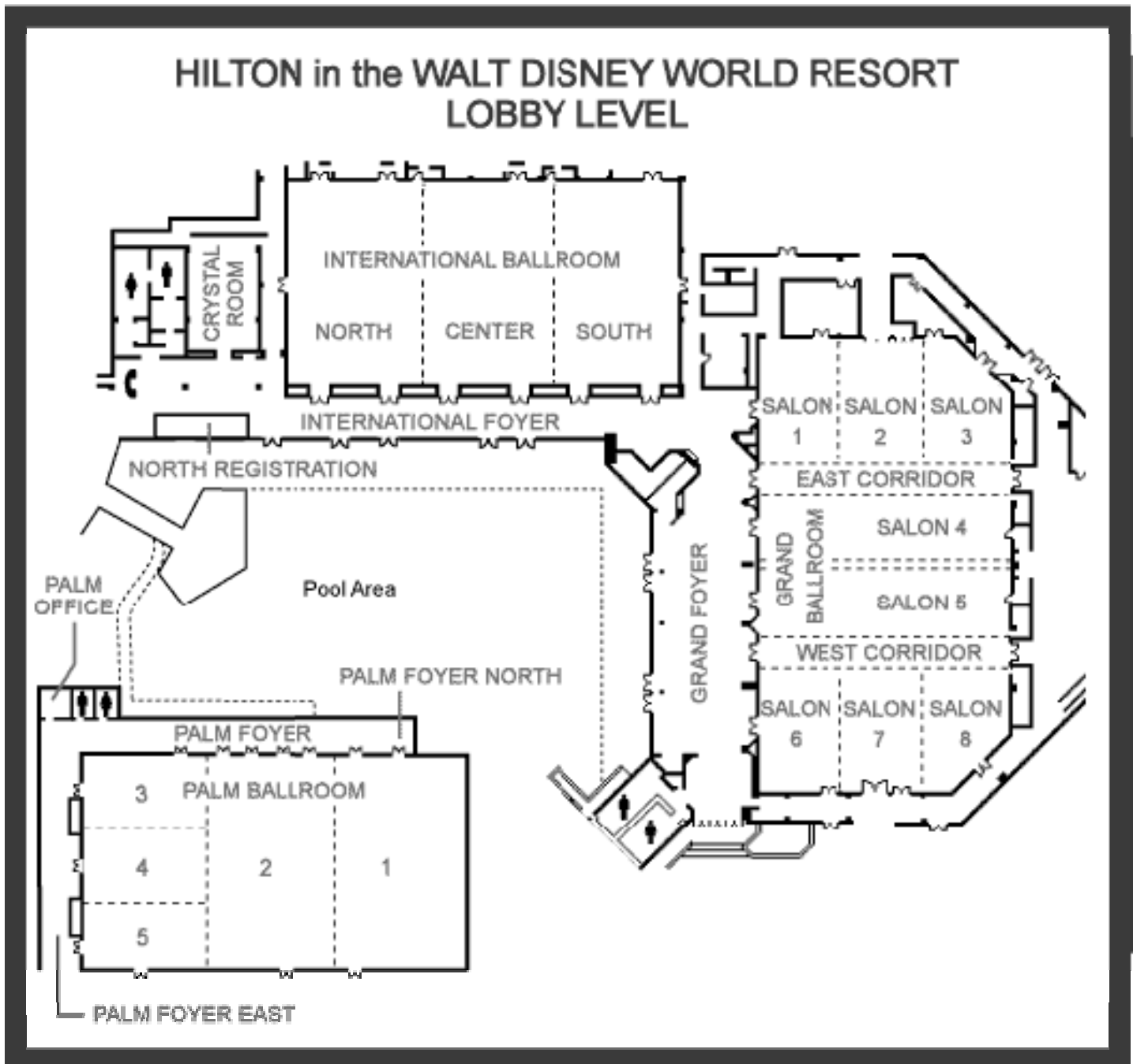


OTHER DINING OPTIONS

Within walking distance of the Hilton WDW at the Downtown Disney Marketplace:

- **Cap'n Jack's Oyster Bar** - Located on the waterfront with windows overlooking the Village lake, this restaurant features seafood specialties. Open for lunch and diner from 11:30 a.m. - 10:30 p.m. New England clam chowder, crab cakes, Chicken Caesar salad, ribs, steaks and Seafood Pasta are among the selections. Price range: Lunch: \$7.99 - \$15.99. Dinner: \$14.99 - \$29.99.
- **Ghirardelli Soda Fountain & Chocolate Shop** - Featuring a wonderful menu of ice cream sundaes, malts, shakes and floats as well as the Ghirardelli line of chocolate. Now offering sugar-free and fat-free ice cream. This popular spot can get extremely busy, particularly in the evening.
- **Fulton's Crab House** - This seafood restaurant is located in the large paddleboat permanently docked at the entrance to Pleasure Island at Downtown Disney. Featured items are lobster, crab, oysters, seasonal specials, steak, combination entrees, chicken and shrimp. **Stone Crab Lounge** - Located in Fulton's, this lounge offers an Oyster bar, oyster sampler, salads, soups and appetizers. Price Range: Lunch - \$8.95 - \$25.95, Dinner - \$17.95 - \$44.95.
- **Portobello Yacht Club** - This wonderful Italian cuisine at Downtown Disney is served in an inviting Mediterranean setting. Indoor and outdoor lakeside seating. Open for lunch and dinner, menu offerings include fresh seafood, wood-burning oven pizza, beef, pasta, chickens and desserts. There's an extensive wine selection. Price Range: Lunch - \$9.00 - \$16.00 / Dinner - \$16.00 - \$45.00.
- **Rainforest Café** - This chain restaurant features animatronic wildlife and a "thunderstorm" that occurs every 22 minutes. The sights and sounds place you in the middle of a tropical rainforest including an amazing saltwater fish tank with vibrant tropical fish, as well as waterfalls and banyan trees. The menu is seafood, steak, sandwiches, salads and pasta. The children's menu includes mac & cheese, burgers and mini-hot dogs. Price Range: \$6.00 (Kid's menu) to \$18.00. There's also a large bar under a 38 foot "Magic Mushroom." It's a great spot to relax and unwind while enjoying one of their specialty drinks.
- **Wolfgang Puck Express** - A "quick service" version of the popular Wolfgang Puck Restaurant at Downtown Disney West Side. Serving breakfast, lunch and dinner. Moderate pricing and a great spot to enjoy an above average counter service meal.
- **Earl of Sandwich** - Freshly prepared sandwiches with specialty sauces and spreads. Salads, wraps, pre-made "Grab 'n Go" selection. Also offering a selection of turnovers, scones, cookies, brownies and ice cream sandwiches.
- Kiosks offering Margaritas, snacks, soft drinks, smoothies, specialty coffee drinks are located throughout the Marketplace

HOTEL FLOORPLAN – LOBBY LEVEL

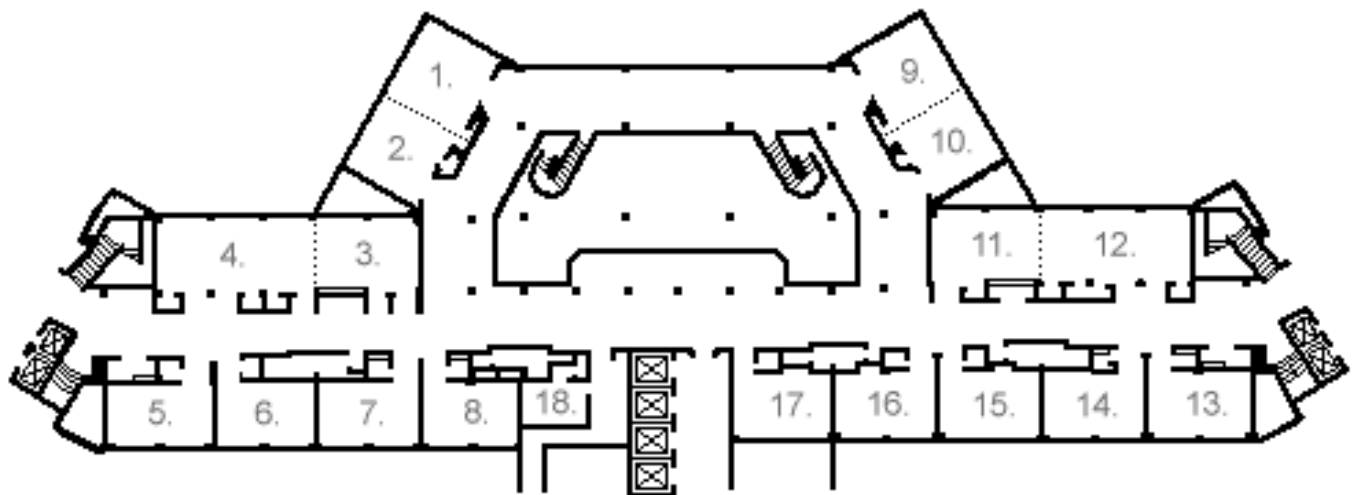


Grand Ballroom – All General Sessions, Breakouts and Open Communications sessions.

International Ballroom – Poster Sessions, Internet Café, Breakfasts & Breaks

HOTEL FLOORPLAN – MEZZANINE LEVEL

HILTON in the WALT DISNEY WORLD Resort - Mezzanine Level




1. QUINCE
2. POINSETTA
3. ORANGE BLOSSOM
4. NARCISSUS
5. MAGNOLIA
6. LILY
7. KAHILI
8. JASMINE
9. AZALEA

10. BEGONIA
11. CAMELIA
12. DOGWOOD
13. EDELWEISS
14. FUSCHIA
15. GARDENIA
16. HIBISCUS
17. IRIS
18. ROSE

SCIENTIFIC PROGRAM

Official Scientific Session support provided by  a Johnson & Johnson company

Sunday, JULY 27 th - Main Arrivals		
12:00 – 2:00 PM	Journal of Neurotrauma Editorial Board Meeting	Grand Ballroom Salon 6
12:00 - 6:00 PM	Registration Desk Open	North Registration Desk
2:00 – 5:00 PM 2.75 CME	SPECIAL SESSION: CLINICAL UPDATE ON MANAGEMENT OF TBI <i>Chair: David Adelson</i> <i>Co-Chair: Rocco Armonda</i> <div style="text-align: right;"><i>Session Support provided by</i> </div> <p>2:00 L1 WHO GETS TBI IN SOUTH FLORIDA - 2:45 <i>Ross Bullock, Univ. of Miami</i></p> <p>2:45 L2 GUIDELINES FOR THE MANAGEMENT OF PEDIATRIC SEVERE TBI - 3:30 <i>P. David Adelson, Univ. of Pittsburgh</i></p> <p>3:30 - 3:45 BREAK</p> <p>3:45 L3 NOVEL THERAPEUTIC INTERVENTIONAL STRATEGIES FOR TBI - 4:30 <i>Andrew Maas, Erasmus</i></p> <p>4:30 CASE PRESENTATION TO THE EXPERTS - 5:00 <i>Bullock, Adelson & Maas</i></p>	Palm Ballroom Salon 3
5:30 - 7:00 PM	STUDENT NETWORKING HAPPY HOUR <i>Meet with other students to network and enjoy poolside refreshments (on your own)</i>	Rum Largo Pool Bar
6:00 PM	EXHIBITOR INSTALLATION & POSTER MOUNTING (Session A)	International Ballroom
7:15 – 9:30 PM	NNS Officers and Councilors Dinner Meeting	Camelia/Dogwood (Mezzanine level)

Monday, JULY 28 th – DAY 1		
7:00 AM - 5:00 PM	REGISTRATION DESK OPEN	North Registration Desk
	NETWORKING CENTER, INTERNET CAFE & EXHIBITS OPEN	International Ballroom
7:00 – 8:00 AM	CONTINENTAL BREAKFAST	International Ballroom & Foyer
8:00 – 8:15 AM	WELCOME & OPENING REMARKS <i>Edward Hall, President, National Neurotrauma Society</i> <i>P. David Adelson, Chair, AANS/CNS Section on Neurotrauma & Critical Care</i>	Grand Ballroom
8:15 – 9:45 1.5 CME	SESSION 1 – CURRENT TOPICS IN SCI REGENERATION <i>Chair: Maria Crowe, Medical College of Wisconsin</i> <i>Co-Chair: John McDonald, Kennedy Krieger Institute</i> <p>8:15 L4 ACTIVITY DEPENDENT PLASTICITY AND TRANSPLANT MEDIATED REPAIR OF THE INJURED SPINAL CORD - 8:45 <i>John Houle, Drexel University</i></p> <p>8:45 L5 ROLE OF GENETIC MODELS IN SCI REGENERATION STUDIES - 9:15 <i>Binhai Zheng, Univ. California San Diego</i></p> <p>9:15 L6 MOLECULAR AND GENETIC MANIPULATION OF EXTRINSIC AXON GUIDANCE - 9:45 FACTORS FOR SPINAL CORD REGENERATION <i>George M. Smith, Univ. Kentucky</i></p>	
9:45 – 11:00	POSTER SESSION A (P1 – P82) & JUDGING SESSION A: TOP STUDENT POSTER COMPETITION (P# 13, 86, 138, 147, 153, 176, 226, 232)	International Ballroom
9:45 AM – 11:00 AM	BREAK & VISIT EXHIBITS	International Ballroom & Foyer

11:00 AM – 12:00 PM	SESSION 2 - OPEN COMMUNICATIONS A (TBI) <i>Chair: Helen Bramlett, Univ. of Miami</i> <i>*Indicates Student Abstract Competition finalist</i> 11:00 P96* - A LINK BETWEEN A NEPRILYSIN GENE POLYMORPHISM AND THE - 11:15 DEVELOPMENT OF AMYLOID- β PLAQUES FOLLOWING TRAUMATIC BRAIN INJURY IN HUMANS <i>Victoria Johnson, Univ. of Pennsylvania</i> 11:15 P191* - BEHAVIOURAL OUTCOME FOLLOWING TRAUMATIC BRAIN INJURY AND - 11:30 SOLUBLE NOGO-66 RECEPTOR THERAPY IN MICE <i>Anders Hanell, Uppsala University, Sweden</i> 11:30 P206* - DEVELOPMENT OF A MODEL OF PRIMARY BLAST BRAIN INJURY: - 11:45 SHOCKWAVES AS A DIRECT CAUSE OF BRAIN INJURY <i>Eugene Park, St. Michael's Hospital, Canada</i> 11:45 P272* - PARP-1 POLYMORPHISMS IN HUMANS AFTER SEVERE TRAUMATIC BRAIN - 12:00 INJURY <i>Ajit Sarnaik, Univ. of Pittsburgh</i>	Grand Ballrooms Salons 1-3
11:00 - 12:00 PM	SESSION 2 – OPEN COMMUNICATIONS B (SCI) <i>Chair: Linda Noble-Hauessein, UCSF</i> <i>*Indicates Student Abstract Competition finalist</i> 11:00 P21* - FUNCTIONAL REGENERATION OF SENSORY AXONS INTO TOPOGRAPHICALLY - 11:15 CORRECT AREAS OF THE SPINAL CORD WITH NEUROTROPHIN TREATMENT IN A MODEL OF BRACHIAL PLEXUS INJURY <i>Pamela Harvey, Tufts University</i> 11:15 P83 - IN VIVO TRACKING OF MESENCHYMAL STEM CELLS IN THE INJURED MOUSE - 11:30 SPINAL CORD <i>Laura Gonzalez-Lara, Robarts Research Institute, Canada</i> 11:30 P290* - LONGITUDINAL STUDIES OF SPINAL CORD CONTUSION INJURY USING - 11:45 MAGNETIC RESONANCE IMAGING <i>Laura Sundberg, Univ. of Texas</i> 11:45 P312 - BIOLOGICAL PROPERTIES OF HYALURONIC ACID IN GLIAL SCAR FORMATION - 12:00 AFTER SCI <i>Zin Khaing, Univ. of Texas at Austin</i>	Grand Ballroom Salons 4-5
11:00 – 1:00 PM	POSTER REMOVAL (SESSION A: P1-P82) POSTER MOUNTNG (SESSION B: P83-P164)	International Ballroom
12:00 - 1:00 PM	FREE TIME FOR LUNCH	<i>On your own</i>
1:00– 2:30 1.5 CME	SESSION 3A – PATHOPHYSIOLOGY OF PEDIATRIC BRAIN INJURY <i>Chair: Christopher Giza, UCLA</i> <i>Co-Chair: George Jallo, Johns Hopkins</i> 1:00 L7 UNDERSTANDING INJURY-SPECIFIC MECHANISMS OF PEDIATRIC BRAIN - 1:30 TRAUMA: ARE WE THERE YET? <i>Ramesh Raghupathi, Drexel University</i> 1:30 L8 METABOLIC DYSFUNCTION AND OXIDATIVE STRESS AFTER TBI IN IMMATURE - 2:00 RATS <i>Courtney Robertson, Univ. of Maryland</i> 2:00 L9 COMPOUNDING BRAIN DEVELOPMENT WITH TBI: WHAT IS GOING ON IN THERE? - 2:30 <i>Mayumi Prins, Univ. California Los Angeles</i>	Grand Ballroom Salons 1-3
1:00– 2:30 1.5 CME	SESSION 3B – CLINICAL UPDATE ON MANAGEMENT OF SPINAL CORD INJURY <i>Chair: Michael Fehlings, U. Toronto</i> <i>Co-Chair: Brian Kwon, U. British Columbia</i> 1:00 L10 HYPOTHERMIA FOR ACUTE TREATMENT OF SCI - 1:30 <i>Barth Green, Univ. of Miami</i> 1:30 L11 BEYOND STEROIDS: UPCOMING TRANSATIONAL THERAPIES FOR SCI - 2:00 <i>Brian Kwon, Univ. British Columbia</i> 2:00 L12 SURGICAL DECOMPRESSION FOR SCI: UPDATE ON THE STASCIS TRIAL - 2:30 <i>Michael Fehlings, Univ. Toronto</i>	Grand Ballroom Salons 4-5
2:30 – 3:45 PM	POSTER SESSION B (P83 – P164) & JUDGING SESSION B: TOP STUDENT POSTER COMPETITION (P# 21, 96, 104, 190, 191, 206, 272, 290)	International Ballroom

2:30 - 3:45 PM	BREAK & VISIT EXHIBITS	International Ballroom & Foyer
3:45 – 5:15 1.5 CME	SESSION 4A – BLAST TBI IN COMBAT CASUALTY CARE AND CIVILIAN TERRORISM <i>Chair: Frank Tortella, WRAIR</i> <i>Co-Chair: Ronald Hayes, Banyan Biomarkers</i> 3:45 L13 TRAUMATIC BRAIN INJURY AND THE GLOBAL WAR ON TERROR - 4:15 <i>Geoffrey Ling, US Army</i> 4:15 L14 PATHOPHYSIOLOGY GUIDED THERAPEUTIC APPROACHES TO BLAST TBI & POLYTRAUMA - 4:45 <i>Patrick Kochanek, Univ. Pittsburgh</i> 4:45 L15 PRECLINICAL MODELS OF BLAST TBI - 5:15 <i>Ibolja Cernak, Johns Hopkins Univ.</i>	Grand Ballroom Salons 1-3
3:45 – 5:15 1.5 CME	SESSION 4B – HANDS-ON WORKSHOP: SPINAL STABILIZATION FOLLOWING TRAUMA <i>Chair: Michael Fehlings, U. Toronto</i> <i>Co-Chair: David Okonkwo</i> <i>Table Instructors: Brian Kwon, James Harrop, Dean Chou, Paul Arnold</i>	Grand Ballroom Salons 4-5
5:15 – 6:00 PM	NNS BUSINESS MEETING <i>All NNS members are invited to attend</i>	Grand Ballroom Salons 1-3
5:45 – 6:45 PM	"WINTRbrainSTORM" BUSINESS MEETING <i>Open to all delegates</i>	Hibiscus/Iris Rooms (Mezzanine level)
5:15 - 6:45 PM	POSTER REMOVAL (SESSION B: P83 – P164) POSTER MOUNTING (SESSION C: P165 – P245)	International Ballroom
6:45 - 8:15 PM	WELCOME RECEPTION <i>All delegates and registered accompanying spouses are invited to attend.</i>	Grand Ballroom Foyer

Tuesday, JULY 29th – DAY 2

7:00 AM - 5:00 PM	REGISTRATION DESK OPEN	North Registration Desk
7:00 AM - 5:00 PM	NETWORKING CENTER, INTERNET CAFÉ & EXHIBITS OPEN	International Ballroom
7:00 – 8:00 AM	CONTINENTAL BREAKFAST	International Ballroom & Foyer
8:00 - 9:30 1.5 CME	SESSION 5 - NUTRITION IN SPINAL CORD & BRAIN INJURY - WHAT SHOULD WE PUT IN THE CHICKEN SOUP? <i>Chair: Patrick Sullivan, U. Kentucky</i> <i>Co-Chair: Ying Deng-Bryant, UCLA</i> 8:00 L16 AUTOPHAGY IN EXPERIMENTAL & CLINICAL CNS INJURY - A NOVEL OF CELL DEATH RELATED TO STARVATION - 8:30 <i>Robert S.B. Clark, U. Pittsburgh</i> 8:30 L17 NUTRITIONAL CONSIDERATIONS IN SCI - 9:00 <i>Wolfram Tetzlaff, U. British Columbia</i> 9:00 L18 METABOLIC MANAGEMENT OF TRAUMATIC BRAIN INJURY: CURRENT STATUS - 9:30 <i>David Hovda, UCLA</i>	Grand Ballroom
9:30– 10:45 AM	POSTER SESSION C (P165 – P245)	International Ballroom
9:30 AM – 10:45 AM	BREAK & VISIT EXHIBITS	International Ballroom & Foyer

10:45 – 1:00 PM	POSTER REMOVAL (SESSION C: P165 – P245) POSTER MOUNTING (SESSION D: P246 – P326)	International Ballroom
10:45 – 11:45 AM 1.0 CME	SESSION 6A - OPEN COMMUNICATIONS (TBI) STUDENT ABSTRACT ORAL PRESENTATIONS <i>Chair: Kathy Saatman, Univ. of Kentucky</i> <i>*Indicates Student Abstract Competition finalist</i>	Grand Ballroom Salons 1-3
	10:45 P86* - THREE-DIMENSIONAL CORTICAL CHANGES IN TRAUMATIC AXONAL INJURY - 11:00 <i>Teddy Youn, Univ. of Texas</i>	
	11:00 P138* - OUTCOME IN BLUNT HEAD TRAUMA PATIENTS WITH GLASGOW COMA - 11:15 SCORE OF THREE ON PRESENTATION <i>Roukoz Chamoun, Baylor College of Medicine</i>	
	11:15 P147* - TREATMENT OF TRAUMA-INDUCED AMPA RECEPTOR MODIFICATION BY - 11:30 PERTURBING PDZ PROTEIN: PROTEIN BINDING <i>Joshua Bell, Univ. of Toronto, Canada</i>	
	11:30 P232* - RESTORATION OF IMPAIRED EXPERIENCE-DEPENDENT PLASTICITY BY - 11:45 ENHANCING NMDA RECEPTOR MEDIATED SYNAPTIC TRANSMISSION AFTER DEVELOPMENTAL TBI <i>Naomi Santa Maria, UCLA</i>	
10:45 – 11:45 AM 1.0 CME	SESSION 6B - OPEN COMMUNICATIONS (SCI) STUDENT ABSTRACT ORAL PRESENTATIONS <i>Chair: Diane Snow, Univ. Kentucky</i> <i>*Indicates Student Abstract Competition finalist</i>	Grand Ballroom Salons 4-5
	10:45 P153* - THE IYs-EGFP-KI MOUSE MODEL REVEALS PREVIOUSLY UNIDENTIFIED - 11:00 POPULATIONS OF HEMATOGENOUS AND MICROGLIAL MACROPHAGES FOLLOWING SPINAL CORD INJURY <i>Sakina Thawer, Univ. of Western Ontario</i>	
	11:00 P176* - MECHANISMS OF THE EFFECT OF HYPERTONIC SALINE VERSUS MANNITOL - 11:15 ON LEUKOCYTE ADHERENCE AND CELL SURVIVAL AFTER TRAUMATIC BRAIN INJURY IN VITRO <i>Donna Watson, Univ. of Miami</i>	
	11:15 P226* - TEMPORAL PROFILE OF MITOCHONDRIAL SUPEROXIDE GENERATION AND - 11:30 APOPTOSIS AFTER MECHANICAL STRETCH IN CORTICAL NEURONS <i>Minke Tang, Univ. of Pittsburgh</i>	
	11:30 P296 - EXAMINATION OF THE EARLY EFFECTS OF NEURAL PRECURSOR CELL - 11:45 TRANSPLANTATION ON THE PERI-LESIONAL ENVIRONMENT OF THE INJURED SPINAL CORD <i>Gregory Hawryluk, Univ. of Toronto</i>	
11:45 - 12:45 PM	FREE TIME / LUNCH <i>*Lunch Items will be available for purchase to take into the WINTR Session</i>	Grand Ballroom East Corridor
11:45 - 12:45 PM	L19 WINTR SPECIAL SESSION: TRANSLATIONAL RESEARCH GRANTS IN NEUROTRAUMA <i>Ramona Hicks Program Director, Extramural Research Program</i> <i>Open to all delegates</i>	Grand Ballroom Salons 1-3
12:45 - 1:00 PM	BREAK - ROOM REFRESH	Grand Ballroom
1:00 – 2:30 PM 1.5 CME	SESSION 7A – NEWER PERSPECTIVES ON ROLE OF OXIDATIVE DAMAGE IN TBI AND SCI <i>Chair: Douglas DeWitt, UTMB</i> <i>Co-Chair: Patrick Kochanek, U. Pittsburgh</i>	Grand Ballroom Salons 1-3
	1:00 L20 GLUTATHIONE PEROXIDASE AS A MODIFIER OF RECOVERY IN THE INJURED, - 1:30 IMMATURE BRAIN <i>Linda Noble-Haeusslein, UCSF</i>	
	1:30 L21 OXIDATIVE LIPIDOMICS IN TRAUMATIC BRAIN INJURY - 2:00 <i>Hulya Bayir, Univ. Pittsburgh</i>	
	2:00 L22 IS INTERRUPTION OF OXIDATIVE DAMAGE ALONE ENOUGH TO ACHIEVE - 2:30 CLINICALLY MEANINGFUL NEUROPROTECTION? <i>Edward Hall, U. Kentucky</i>	

1:00 – 2:30 PM 1.5 CME	SESSION 7B - SURGICAL DECOMPRESSION IN TBI <i>Chair: Geoffrey Manley, UCSF</i> <i>Co-Chair: Dilantha Ellegala, Med. Univ. South Carolina</i> 1:00 L23 SURGICAL DECOMPRESSION OF CEREBRAL CONTUSIONS – 1:30 <i>Takeshi Maeda, Tokyo Univ.</i> 1:30 L24 DECOMPRESSIVE CRANIECTOMY FOR INTERCRANIAL HYPERTENSION – 2:00 <i>Bizhan Aarabi, Univ. Maryland</i> 2:00 L25 SURGICAL DECOMPRESSION FOR PENETRATING TBI – 2:30 <i>James Ecklund, Inova Fairfax Hospital</i>	Grand Ballroom Salons 4-5
2:15 – 3:30 PM	POSTER SESSION D (P246 – P326)	International Ballroom
2:15 - 3:30 PM	BREAK & VISIT EXHIBITS	International Ballroom & Foyer
3:30– 5:00 PM 1.5 CME	SESSION 8A – GLIAL CELL RESPONSES AND INTERACTIONS IN CNS INJURY <i>Chair: Candace Floyd, U. Alabama</i> <i>Co-Chair: Bruce Lyeth, UC Davis</i> 3:30 L26 MODULATING MICROGLIAL ACTIVITY AFTER BRAIN OR SPINAL CORD TRAUMA – 4:00 <i>Alan Faden, Georgetown Univ.</i> 4:00 L27 OLIGODENDROGLIAL DEGENERATION AND REPLACEMENT STRATEGIES IN SCI – 4:30 <i>Jeffrey Kocsis, Yale University</i> 4:30 L28 ROLES AND REGULATION OF REACTIVE ASTROGLIA IN SPINAL CORD INJURY – 5:00 <i>Michael Sofroniew, UCLA</i>	Grand Ballroom Salons 1-3
3:30– 5:00 1.5 CME	SESSION 8B - ICU MANAGEMENT AND ADVANCED NEUROMONITORING <i>Chair: Geoffrey Manley, UCSF</i> <i>Co-Chair: Ross Bullock, U. Miami</i> 3:30 L29 SERUM AND CSF BIOMARKERS IN THE NEUROICU – 4:00 <i>Claudia Robertson, Baylor University</i> 4:00 L30 ADVANCED BIOINFORMATICS IN THE NEUROICU – 4:30 <i>Geoff Manley, Univ. California San Francisco</i> 4:30 L31 BRAIN OXYGEN MONITORING FOR NEUROTRAUMA OR CONTINUOUS EEG FOR – 5:00 NEUROMONITORING FOLLOWING TBI ICU MONITORING FOR NEUROTRAUMA <i>Paul Vespa, Univ. California Los Angeles</i>	Grand Ballroom Salons 4-5
3:30 – 6:00 PM	POSTER REMOVAL (SESSION D)	International Ballroom
5:00 – 7:00 PM	WINTR RECEPTION & MENTORING PROGRAM <i>* Advance purchase ticket required *</i>	Palm Ballroom Salons 3-5

Wednesday, July 30th – Day 3

7:00 AM - 1:00 PM	REGISTRATION DESK OPEN	North Registration Desk
	NETWORKING CENTER, INTERNET CAFÉ & EXHIBITS OPEN	International Ballroom
7:00 – 8:00 AM	CONTINENTAL BREAKFAST	International Ballroom & Foyer
8:00 - 9:30 AM 1.5 CME	SESSION 9 – MILD TRAUMATIC BRAIN INJURY <i>Chair: Thomas Reeves, Virginia Commonwealth Univ.</i> <i>Co-Chair: Jonathan Lifshitz, U. Kentucky</i> 8:00 L32 MILD TBI-CLINICAL OVERVIEW – 8:30 <i>Mark Lovell, UPMC</i> 8:30 L33 "MILD TRAUMATIC BRAIN INJURY" IS AN OXYMORON – 9:00 <i>Douglas H. Smith, Univ. Pennsylvania</i> 9:00 L34 MILD TBI IN MILITARY PERSONNEL – 9:30 <i>Charles Hoge, Walter Reed Army Inst. Research</i>	Grand Ballroom
9:30 - 10:30 AM	BREAK & VISIT EXHIBITS	International Ballroom & Foyer

10:30-11:30 AM 1.0 CME	SESSION 10 – NEW NIH INITIATIVES IN TBI RESEARCH <i>Chair: Ramona Hicks, NIH/NINDS</i> <i>Co-Chair: Jean Wrathall, Georgetown Univ.</i> 10:30 L35 THE NIH EFFORT TO DEVELOP A NEW TBI CLASSIFICATION TO FACILITATE -11:00 THERAPEUTIC TRANSLATION <i>Geoffrey Manley, UCSF</i> 11:00 L36 COMBINATION THERAPIES FOR TBI -11:30 <i>Ramona Hicks, NIH/NINDS</i>	Grand Ballroom
11:30 – 12:00 PM	STUDENT POSTER COMPETITION AWARDS CEREMONY & CLOSING REMARKS	Grand Ballroom
1:00 PM	REGISTRATION/INFORMATION DESK CLOSES	North Registration Desk
12:00 – 3:00	NNS/INTS 2009 PLANNING MEETING & LUNCH	Crystal Room

FACULTY PRESENTATION ABSTRACTS

L1

WHO GETS TBI IN SOUTH FLORIDA?

Ross Bullock, Univ. of Miami

The Ryder Trauma Center is the only Level 1 trauma center for about 3.5 million people in the three counties of South Florida. We compared census records and the RTC registry to determine the demographic profile of traumatic brain injury (TBI) in our region. **Results** - Admission for severe and moderate TBI over the years 2005, 2006, 2007 averaged 800 per year, 75% male and 7.4% of injuries were due to gun shot wound/penetrating brain injury. Thirteen percent required craniotomy for hematoma removal or ICP control. Although Hispanics comprise 61% of the population of Miami-Dade County, they represented only 34% of the TBI, while Whites represent 18% of the population but 43% of TBI. African-Americans represent 20% of the population, and represented 22.5% of TBI admissions. Over 50% of admissions were in the 18-44 age groups, and only 15% of TBI were seen in over 65 year olds. **Conclusion** - In South Florida, severe and moderate TBI is seen most commonly in young, White (non-Hispanic), males, and the demographic pattern is similar to that elsewhere in the United States.

L2

GUIDELINES FOR THE MANAGEMENT OF PEDIATRIC SEVERE TBI

P. David Adelson, Univ. Pittsburgh

Since the publication of the evidence-based *Guidelines for the Acute Medical Management of Severe Traumatic Brain Injury in Infants, Children, and Adolescents* (Adelson et al, *PCCM*, 2003), there has been an increasing focus on the unique pathophysiological and neurobiological distinctions associated with TBI in children. While specific mechanisms in different ages and geographic regions may vary, TBI in the young constitutes a significant medical and socioeconomic problem worldwide. Young persons who survive moderate-severe TBI may be left with both overt and subtle neurological and cognitive impairments and bear the burden of these deficits life-long. Additionally, unique to children is the group that suffers inflicted TBI (non-accidental trauma) as a major cause of TBI in infants, is associated with repeated, often severe diffuse injury, and has elements of associated hypoxic injury. It is often slow to be diagnosed and the outcome in these patients is generally worse than other types of TBI. While there remain significant shortcomings in management of pediatric patients, with no specific standards of care, and recommendations mostly derived from treatment of the adult population, aggressive treatment particularly with ICP monitoring has improved outcomes in children following TBI. Future treatments will focus on reducing secondary injury unique to the pediatric brain. Understanding the maturational distinctions is crucial to improved patient care and to the development of therapeutic interventions that may uniquely benefit this age range, such as hypothermia, decompressive craniectomy and age-specific pharmacotherapies. This talk will highlight the unique aspects of pediatric TBI and its management both the present and the potential directions for future novel interventions.

Objectives:

- 1) The participant will be able to understand the basics of evidence based documents and guidelines including classes of evidence and levels of recommendations
- 2) The participant will be able to understand the concepts of primary injury, second insults, and secondary injury and their impact on outcome in pediatric traumatic brain injury.
- 3) The participant will be able to understand the management algorithm for the clinical management

of children with severe traumatic brain injury for immediate implementation into practice.

L3

NOVEL THERAPEUTIC INTERVENTIONAL STRATEGIES FOR TBI

Andrew Maas, Erasmus

Many randomized controlled trials (RCT's) have been performed to investigate the effectiveness of new therapies in traumatic brain injury (TBI), but none have convincingly shown benefit. Clinical trials in TBI pose complex methodological challenges related to the heterogeneity of the disease. First, TBI is not one single disease entity but includes a complex spectrum of pathologies and uncertainty exists as to whether pathophysiologic processes targeted are indeed active in individual patients and if so at what time after injury. Much basic and clinical research is needed before this utopian goal can ever become clinically realistic. Second, patients included in trials are heterogeneous in terms of clinical severity and baseline prognostic risk. It is here, that methodologic approaches may be optimized to increase statistical power. The aim of our studies is to optimize the design and analysis of clinical trials in TBI with the expectation of increasing the likelihood of demonstrating the benefit of a truly effective new therapy or therapeutic agent in victims of a head injury.

We have created the IMPACT database on TBI through merging individual patient data from 8 RCT's and 3 observational surveys including individual patient data on 9205 subjects. The IMPACT database will be expanded as our studies continue. This database forms a culture medium in which innovative approaches to improving trial design and analysis are being explored. In simulation studies we investigated benefits of adjusting for heterogeneity with covariate adjustment, by exploiting the ordinal nature of the Glasgow Outcome Scale and by relating the outcome obtained in individual patients to their baseline prognostic risk. Extensive prognostic analysis was performed to identify appropriate covariates and to develop and validate prognostic models necessary to establishing the baseline prognostic risk.

Most prognostic information was contained in a set of 7 covariates: age, motor score, pupillary reactivity, CT classification, traumatic subarachnoid hemorrhage, hypoxia and hypotension. We successfully developed and validated prognostic models of increasing complexity. Simulation studies showed that adjustment for the 7 main covariates yielded a potential reduction in sample size of about 25%. Exploiting the ordinal nature of the GOS in a proportional odds analysis yielded a sample size reduction of approximately 30% in comparison to analyses based on the traditional dichotomy of the GOS into unfavorable versus favorable outcome. Differentiating the outcome analysis to the baseline prognostic risk (sliding dichotomy) showed a potential sample size reduction of approximately 35%. Combining covariate adjustment with proportional odds analysis or the sliding dichotomy approach showed a potential to increase statistical power by 50%.

Covariate adjustment and novel approaches towards outcome analysis have the potential to increase statistical power in TBI trials by approximately 50%.

Objectives:

1. Describe the appropriate approach to prove clinical benefit of novel treatments in TBI.
2. Define and discuss promising intervention strategies for treatment of TBI.
3. Outline preferred methods for dealing with the inherent heterogeneity of the population of TBI patients included in clinical trials.

L4**ACTIVITY DEPENDENT PLASTICITY AND TRANSPLANT MEDIATED REPAIR OF THE INJURED SPINAL CORD**

John Houle, Drexel University

Spinal cord injury is a complex and evolving condition that likely will require a combination of therapeutic approaches applied at different post-injury intervals to address the myriad of problems that arise during acute and chronic stages of the injury. Studies in our group are focused on exploring neuroprotective, neuroregenerative and rehabilitation approaches for a variety of spinal cord injury models. For example, recent use of peripheral nerve grafting techniques combined with enzyme digestion of inhibitory extracellular matrix components within the glial scar demonstrated the potential to enhance axonal regeneration beyond an injury site, with some measure of associated functional recovery. With a different approach our use of forced fore- and hind limb exercise paradigms promotes an intraspinal increase in the expression of neurotrophic factors and change in the pattern of cytokine expression that may be conducive to neuroprotection. However, such activity-dependent plasticity has not led to significant improvement in locomotor behavior, suggesting the need for additional therapeutic interventions to support recovery. This presentation will highlight our efforts to advance the combined use of transplantation and exercise approaches as we attempt to develop a treatment strategy that can be translated to future clinical application.

Objectives:

- 1) Recognize that a successful approach to treating spinal cord injury will require attention to upstream and downstream areas affected by the injury in addition to intervention at the site of injury.
- 2) Identify the advantages to therapeutic approaches that address both short and long axon tracts as sources for functional regeneration.
- 3) Discuss the potential beneficial actions of physical rehabilitation after experimental spinal cord injury, with particular regard to the role of exercise-induced neurotrophic factor production.
- 4) Discuss the importance of a combination treatment strategy for the promotion of repair after spinal cord injury.

L5**ROLE OF GENETIC MOUSE MODELS IN SCI REGENERATION STUDIES**

Binhai Zheng, Univ. of California, San Diego

Therapeutic development for spinal cord injury will benefit significantly from a better understanding of the molecular mechanisms of axon regeneration failure in the adult CNS. Genetically modified mice have gained increased utility in axon regeneration research, particularly with models of spinal cord injury. Genetic mouse models have been used to assess the contribution of known neurite growth inhibitors in regeneration failure, to identify new candidate inhibitors of regeneration, and to gain molecular insight on other aspects of spinal cord injury, recovery and repair such as astroglial response. In vivo imaging of injured spinal axons in mice has advantages over conventional endpoint analysis in examining axon degeneration and regeneration. Together, studies with mouse genetic models are providing unique and valuable insights on SCI and axon regeneration.

Objectives:

1. Discuss the advantages of using the laboratory mouse as a model organism to study experimental SCI, especially regarding axon regeneration
2. Distinguish different methodologies to genetically modify mice and identify the utility of each

3. Discuss the significance of results obtained with genetic mouse models and assess the impact on drug target validation and pre-clinical development
4. Discuss in vivo imaging as a specific example of utilizing genetically modified mice to study axon regeneration
5. Recognize the disadvantages of genetic mouse models and describe ways to overcome them

L6**MOLECULAR AND GENETIC MANIPULATION OF EXTRINSIC AXON GUIDANCE FACTORS FOR CNS REPAIR AND REGENERATION**

George M. Smith, Univ. of Kentucky

Implicit to functional recovery after CNS injury is re-establishment of appropriately organized circuits. During development, a plethora of guidance molecules, both attractive and repulsive, are expressed in a temporally and spatially organized manner to direct axon growth and circuit formation. With cessation of development many of these guidance pathways are turned off, and injury to the adult CNS can cause further disruption in expression patterns, particularly at the lesion site. Using viral mediated gene expression, we are able to create guidance pathways to target regenerating sensory axons or direct the growth of axon from neuronal transplants. In the former model, slightly overlapping gradients of nerve growth factor (NGF) and semaphorin 3A were expressed in the dorsal horn to induce laminar specific regeneration of nociceptive axons. In the other model, pathways were generated extending across the corpus callosum and turning into the contralateral striatum. With expression of NGF and fibroblast growth factor-2, sensory axons grew along the corpus callosum, across the midline, and toward an NGF-expressing target in either the contralateral striatum: a distance of 7– 8 mm including a 90° turn from white matter into gray matter. Furthermore, expression of semaphorin 3A slightly dorsal and lateral to the turning point increased the number of axons turning into the striatal target. Likewise, similar pathways of glial cell line-derived neurotrophic factor (GDNF) and GDNF family receptor (GFR- 1) induced growth of axons from E14 ventral mesencephalic dopaminergic neuron transplants to follow the pathway and extend into the contralateral striatum. We are presently examining similar pathways to reconstruct the nigrostriatal pathway in a rodent Parkinson's disease model. These results show that judicious expression of neuron-specific chemoattractant and chemorepellant molecules using viral vectors can be used to target axon regeneration or the growth of axons from neuronal transplants in the adult CNS. Supported by NINDS R01 NS38126 and the Kentucky Spinal Cord and Head Injury Research Trust #6-13.

Objectives:

- 1) Discuss the advantages of using viral gene therapy to establish growth supportive pathways and gradients within the adult CNS.
- 2) Identify the advantages for using guidance molecules to "recapitulate" a developmental-like program.
- 3) Discuss advantages of targeting axon regeneration to aid reformation of laminar specific circuits.
- 4) Discuss application of guidance pathways in long distance growth of axons from transplanted neurons and its potential in circuit reconstruction.

L7**UNDERSTANDING INJURY-SPECIFIC MECHANISMS OF PEDIATRIC BRAIN TRAUMA: ARE WE THERE YET?**

Ramesh Raghupathi, Drexel University

The mechanisms underlying the pathophysiology of traumatic brain injury (TBI) in the adult population have been intensively studied over the past 4 decades. In the same period, the less-than-optimal attention to pediatric TBI may be due to certain assumptions about the immature brain with regards to the pathologic alterations after trauma (similar to that in adults) or its response to trauma (enhanced recovery). More recently, with the

development of age-appropriate animal models of pediatric TBI, these assumptions have largely proven to be inaccurate. In addition to age, the mechanism of injury is an important parameter that needs to be considered in the evaluation of a clinically-relevant animal model of pediatric TBI. Children less than 4 years of age appear to be more vulnerable – higher mortality and greater morbidity – to TBI than either older children or young adults; survivors are faced with a lifetime of behavioral and functional deficits. The neuropathologic basis for the functional deficits appears to be a combination of diffuse (traumatic) axonal injury and contusions. In order to investigate the biochemical mechanisms underlying the neuropathologic alterations and thereby develop appropriate treatment strategies, it is necessary to utilize injury-specific animal models. To this end, we have developed an animal model of diffuse (non-contusive) brain trauma and one of contusive trauma in the 17-day-old rat, an age that is neurologically equivalent to a toddler. Similar to that in adult animals, diffuse brain injury in the immature rat is characterized by widespread axonal damage with unique injured axon populations demonstrating either impaired axonal transport or neurofilament compaction. Interestingly, the immunophilin FK506 appears to selectively inhibit NFC but does not affect the extent of impaired axonal transport (IAT), data that are contrary to the observations in adult animals subjected to diffuse brain trauma. Contusive brain trauma results in calpain and caspase-3 activation in and around the contusion site, in a pattern that is similar to that observed in adult animals. However, glutamate receptor antagonists administered in the acute period exacerbate the extent of calpain and caspase-3 activation, an observation at odds with that in adult animals. These data underscore the importance of utilizing age-appropriate and injury-specific models of pediatric TBI. (Supported by NIH grants NS41561 and NS053651)

Objectives:

- 1) Define the pathologic and behavioral alterations following pediatric brain trauma
- 2) Identify the similarities and differences between contusive and diffuse brain trauma in the immature animal
- 3) Discuss mechanisms underlying the differential pathophysiology of contusive and diffuse brain trauma
- 4) Discuss the utility of injury-specific acute therapeutic strategies in the immature animal

L8 METABOLIC DYSFUNCTION AND OXIDATIVE STRESS AFTER TBI IN IMMATURE RATS

Courtney Robertson, Univ. of Maryland

It is well known that free radical production and oxidative stress are primary contributors to neurologic injury in adult TBI. A growing number of preclinical and clinical studies in pediatric TBI have shown evidence for significant oxidative stress. Clinical studies of CSF from children after severe TBI have shown increased markers of protein oxidation and lipid peroxidation, with depletion of antioxidants. Detoxification of free radicals in the brain is dependent on a number of antioxidant enzymes (e.g., superoxide dismutase, glutathione peroxidase) and low molecular weight antioxidants (e.g., glutathione, ascorbate). The endogenous, baseline levels of both the antioxidant enzymes and their supportive antioxidant molecules change throughout development, with the majority being lower in young brains compared to the adult brain. In addition to lower baseline levels, the developing brain may have a blunted antioxidant response to acute injury than the adult brain. It is well established that free radicals can directly contribute to post-injury mitochondrial damage, which can cause devastating alterations in mitochondrial bioenergetics following TBI, ultimately leading to complete energy failure and cell death. This means that effective antioxidant strategies that will limit these diverse alterations in cerebral energy metabolism will need to target multiple pathways of both cellular and mitochondrial ROS production and detoxification, in order to have overall improvements in cerebral energy metabolism. Furthermore, these neuroprotective interventions

will need to focus on age-specific aspects of the response to and recovery from acute TBI.

Objectives:

1. Identify the alterations in cerebral metabolism that occur following TBI in the developing brain.
2. Discuss the significance of oxidative stress after TBI and identify the potential vulnerability of the immature brain to free radical injury.
3. Outline the impact of oxidative stress on brain metabolism in pediatric TBI, and identify potential neuroprotective approaches based on this pathology.

L9 COMPOUNDING BRAIN DEVELOPMENT WITH TBI: WHAT IS GOING ON IN THERE?

Mayumi Prins, Univ. of California, Los Angeles

During cerebral development there are numerous “systems” that are simultaneously maturing at their programmed rates. The progression of these maturing systems can be violently disrupted, altered or destroyed when brain development is compounded with traumatic brain injury (TBI). The antioxidant machinery, cerebral metabolism, the immune system and plasticity potentials are examples of systems that have been recently studied after developmental TBI. Differences in maturity of antioxidant systems are manifest as the failure of the younger brain to increase glutathione peroxidase activity after TBI. The ability of the developing brain to combat oxidative stress contributes to their different metabolic responses. Normal maturation of brain metabolism involves substrate switching. After TBI, the processing of glucose is altered and administration of alternative substrates has been shown to improve outcome. In contrast to the immature antioxidant system, the immune response of the younger brain has been shown to be greater than adults. After TBI, a ‘hyperactive’ inflammatory response may be detrimental and this may be an age-specific target for treatment. Finally, the developing brain is well known for its greater potential for plasticity. With ongoing axonal growth, innervation, synapse formation and experience-dependent activation, is the younger brain better “primed” for rewiring after TBI? Evidence of axonal injury, cell death and denervation post-injury has induced research interest in molecules associated with growth (BDNF, IGF), synaptic proteins, and synaptic function (NMDA receptors). These studies have provided insight into mechanism of TBI-induced damage and the promise of enhanced recovery. It is essential to understand the normal progression of various systems in cerebral development in order to better comprehend the age-specific consequences of TBI and as well as novel potential therapeutic opportunities. A multi-system approach shows great promise for unlocking developmental mechanisms of injury response and recovery.

Objectives:

- 1) Examine how traumatic brain injury affects ongoing cerebral developmental changes, specifically in regards to measures of metabolism and plasticity
- 2) Understand the compound effect of these changes has brought some insight into possible therapeutic approaches including alternative substrates and NMDA stimulation.

L10 HYPOTHERMIA FOR ACUTE TREATMENT OF SCI

Barth Green, Univ. of Miami

No abstract received prior to publication.

Objectives:

L11

BEYOND STEROIDS: UPCOMING TRANSLATIONAL THERAPIES FOR SCI

Brian Kwon, Univ. British Columbia

While controversy continues to exist about the therapeutic benefit of methylprednisolone administration in acute SCI, there is little debate about the urgent need for more convincingly efficacious neuroprotective and neuro-reparative agents. This lecture will provide an overview of the biological rationale for a variety of novel neuroprotective interventions and the progress that has been made in their clinical evaluation. Such interventions include minocycline, Cethrin, and the anti-Nogo antibody. Challenges surrounding the clinical evaluation of and translation of novel therapeutic agents will also be discussed.

Objectives:

- 1) Review basic pathophysiologic mechanisms that contribute to secondary damage after acute SCI
- 2) Discuss clinical trials of acute neuroprotective or neuro-reparative agents that have either begun or will begin in the near future.
- 3) Analyze the pitfalls in the interpretation of media reports of acute and chronic SCI therapies, and early non-randomized clinical trials.

L12 SURGICAL DECOMPRESSION FOR SPINAL CORD INJURY: "UPDATE ON THE STASCIS TRIAL"

Michael G. Fehlings MD, University of Toronto

The role and timing of surgical intervention of acute spinal cord injury remains highly controversial. Despite compelling biological evidence to suggest that early decompression of the injured spinal cord results in neurological recovery, surgeons have been reluctant to undertake such an approach due to concerns related to increased medical and neurological complications. However emerging evidence indicates that with marked improvements in imaging, spinal surgical techniques and neuro anesthetic protocols early decompression can be undertaken in a safe and efficacious manner.

Objectives:

1. Gain an understanding of the biological/clinical rationale for decompression of the injured spinal cord.
2. Learn about the outcomes of the STASCIS clinical trial including clinical implications; impact on neurological recovery and complications.

L13 TRAUMATIC BRAIN INJURY AND THE GLOBAL WAR ON TERROR

Geoffrey S. F. Ling, M.D., Ph.D., Colonel, Medical Corps, U.S. Army

Traumatic brain injury (TBI) has always been a significant disease state for the military, even in peacetime. However, when war erupts, the etiology changes from accidents and motor vehicle collisions to fragments, bullets and explosions. Military medical providers use clinical management guidelines and aggressive neurocritical care that were developed for civilian care. This has contributed to improved survival and outcome. However, the lack of a specific TBI therapy has limited the impact of these advances. For the military, there are unique aspects of combat related TBI. An obvious difference from the civilian experience is the prevalence of penetrating injuries, mainly by low velocity high mass fragments. Also, there is now the recognition that explosive blast may result from mechanisms that are distinct from acceleration-deceleration or focal concussive impact. From a strictly clinical standpoint, severe explosive blast TBI has unusual features such as early hyperemia, rapid malignant edema, high prevalence of subarachnoid hemorrhage and vasospasm. Mild explosive blast TBI is an emerging clinical condition. It has proven difficult to diagnose and clinically treat. Major efforts are

underway to ameliorate this. The overlap with post-traumatic stress disorders is important and needs explanation. All of this underscores that combat related TBI is a disease where more research is needed but research can and will provide important insights.

Objectives:

1. Discuss military TBI
2. Review unique aspects of explosive blast TBI
3. Review ongoing DARPA funded TBI initiatives relevant to TBI

L14 PATHOPHYSIOLOGY GUIDED THERAPEUTIC APPROACHES TO BLAST-TBI AND POLYTRAUMA

Patrick Kochanek, Univ. of Pittsburgh

Acute therapeutic approaches to blast-traumatic brain injury (TBI) should target key aspects of a complex secondary injury cascade. Five factors must be considered, 1) facets of TBI that are common across injuries, 2) unique pathophysiological features of blast-TBI, 3) complicating extracerebral insults (shock/polytrauma), 4) insult severity, and 5) dose response and brain pharmacodynamics/kinetics. We must account for temporal factors that influence the use of treatments in the field, emergency department, operating room, and ICU. Conventional facets of TBI that are targets include neuronal death, excitotoxicity, edema, axonal injury, oxidative stress, mitochondrial damage, ischemia, and inflammation, synaptic injury, and disturbances in cell signaling, among others. Much remains to be discovered, but early data suggest that several aspects of TBI pathophysiology are of special importance in blast-TBI—particularly after severe injury—including malignant edema, vasospasm, axonal injury, and intracerebral hemorrhage. New therapies will be superimposed on current treatment, which for severe blast-induced TBI is substantial. Two approaches to treat blast-TBI will be discussed 1) therapies shown to have promise in conventional TBI models or phase I-II clinical trials. These “low-hanging fruit” should be prioritized taking into consideration the unique pathophysiology of blast-TBI, and tested in emerging models of blast-TBI, 2) more speculative, but potentially higher yield therapies should be explored via high-throughput screening, in rodent models of conventional or blast TBI (across injury levels, gender, and +/- shock/polytrauma). And the most promising agents advanced to large animal models of blast-TBI/clinical trials. There may be many shared mechanisms across injury levels in blast-TBI, but optimal treatment for mild and severe blast-TBI likely differ. Novel therapies should also be explored including agents that target subcellular compartments (ie mitochondria), new delivery systems (ie nanoparticles), cellular therapies, and new hemoglobin-based resuscitation fluids. The most promising therapies should be tested in models that include standard care. Combined therapies are also likely to be needed. Finally, acute therapies should set the stage for optimal rehabilitation/regeneration in a continuum of care from the field to rehabilitation. Support: DARPA PREVENT, US Army PR054755 W81XWH-06-10247, NS38087, NS30318

Objectives:

- 1) Recognize the complex effects of polytrauma in the setting of both experimental and clinical traumatic brain injury.
- 2) Familiarize yourself with experimental models of combined traumatic brain injury and secondary insults – including blast-induced traumatic brain injury.
- 3) Understand some of the key features of extracerebral and cerebral effects of blast injury.
- 4) Recognize the important role of blast-induced traumatic brain injury in both the setting of combat casualty care and civilian terrorist attacks.

L12 PRECLINICAL MODELS OF BLAST-INDUCED NEUROTRAUMA

Ibolja Cernak, Johns Hopkins University

The increased use of improvised explosive devices (IEDs) in military and terrorist scenarios is a growing threat to military and civilian populations. Primary, secondary, tertiary, and quaternary blast effects cause complex injuries, often involving multiple organs and organ systems. These injuries may manifest immediately (acute) or develop later (latent). Blast-induced neurotrauma (BINT) is among most debilitating long-term consequences of a blast exposure, and poses special challenges because the initial symptoms are often hidden and may fully develop months or even years after blast exposure. People with BINT can show memory loss for events before and after the explosion, confusion, headache, impaired sense of reality, and reduced decision-making capacity. The timely recognition of BINT is necessary for proper triaging, evacuation, and monitoring of casualties for neurological complications. Adequate diagnosis of acute BINT and appropriate treatments of injuries such as brain edema or cerebral vasospasm could save lives. The prompt initiation of rehabilitative measures at the first symptoms of chronic BINT could reduce long-term neurological disability.

Objectives:

- 1) Outline the importance of blast-induced neurotrauma and describe related clinical findings;
- 2) Define and discuss the pathobiology of blast-induced neurotrauma, and differentiate primary, secondary, tertiary, and quaternary blast-induced neurological consequences;
- 3) Describe the currently available preclinical models of blast-induced neurotrauma, and discuss the pros and cons of these models;
- 4) Discuss the most important research directions concerning the diagnosis, monitoring, treating and mitigating blast-induced neurotrauma.

L13

AUTOPHAGY AFTER BRAIN INJURY: FRIEND, FOE, OR BYSTANDER?

Robert S.B. Clark, Univ. of Pittsburgh

Autophagy is an important homeostatic process for recycling of proteins and organelles. We, and others, have recently shown that autophagy is increased after traumatic brain injury (TBI); however, its role after traumatic and other types of acute brain injury remains undefined. In contrast to a protective role for autophagy in chronic neurodegenerative diseases associated with abnormal accumulation of proteins ("amyloidopathies"), autophagy appears to play a detrimental role after acute cerebral ischemia. Whether autophagy is beneficial, detrimental, or neither likely depends upon the mechanism of injury and bioenergetic state of the brain. Using in vitro and in vivo models, we are striving to establish the role for autophagy after TBI, and determine whether manipulating autophagy represents a clinically relevant therapeutic modality for the treatment of acute brain injury.

Objectives:

1. Review the physiologic process of autophagy as it relates to brain injury.
2. Demonstrate sex-dependent differences in autophagy.
3. Provide evidence that autophagy is increased after traumatic brain injury.
4. Explore the potential role(s) of autophagy after traumatic and other types of acute brain injury.

L14

NUTRITIONAL CONSIDERATIONS IN SCI – WHAT THE ANIMAL MODELS TEACH US

Wolfram Tetzlaff, Univ. of British Columbia

There are no evidence-based clinical guidelines for nutrition after spinal cord injury. We discovered in rats that every-other-day-fasting (EODF) starting after an incomplete cervical spinal cord injury (SCI), improved functional recovery in several behavioral tests. Hence, we coined the term therapeutic EODF. Histologically,

EODF treated animals showed a dramatic reduction in lesion size and enhanced sprouting of the corticospinal tract. The precise mechanisms are unknown. EODF treatment lowered fasting blood glucose levels and increased beta-hydroxybutyrate levels which is known to exert neuroprotective effects. Interestingly, a dampened cellular inflammatory response, and an enhanced astrocyte alignment at the lesion surface in the EODF animals on day 5 might have contributed to the neuroprotective mechanisms. Young adult male rats on this regimen still gain weight, albeit at a slower rate, and consume 150-160% of their normal daily calories on the eating days. We subsequently asked whether this effect can be found in more than one model of SCI. To mimic a more clinically relevant injury mechanism, a moderate low thoracic contusion injury was induced in adult SD rats. Behaviorally, animals treated with EODF started before or after thoracic injury reached higher open field locomotion scores compared to ad libitum fed animals or pair-fed animals. In addition, when analyzing the gait pattern using the Catwalk system, both EODF groups (started before and after the injury) performed better than the ad libitum fed and pair fed control groups. The histopathological analysis is still under way and possible mechanisms will be discussed. In conclusion, therapeutic EODF is a simple regimen that displays robust, albeit moderate, effects in two different spinal cord injury paradigms in rats making it an interesting candidate for clinical translation. We also propose that this treatment may provide benefits to prevent metabolic syndrome and cardiovascular disease in the chronic state of spinal cord injury. Dietary restriction, both caloric restriction and intermittent fasting, have been proven to be beneficial in a wide variety of animal models for chronic diseases including diabetes and cardiovascular disorders.

Supported by Craig H. Neilsen Foundation, Christopher and Dana Reeve Foundation and CIHR of Canada.

Objectives:

- 1) Understand the nutritional guidelines for patients with acute spinal cord injury are not evidence-based
- 2) Discuss how improved behavioral outcomes after incomplete cervical and thoracic spinal cord injury were observed if the rats were subjected to an intermittent fasting regimen beginning at the time of injury
- 3) Learn how intermittently fasting rats showed smaller lesions, less inflammation and increased neural sprouting.
- 4) Analyze how nutritional regimen may have impact on outcome in patients

L15

METABOLIC MANAGEMENT OF TRAUMATIC BRAIN INJURY: CURRENT STATUS AND EMERGING CONCEPTS

David Hovda, UCLA

Glucose is traditionally considered the primary, if not sole, fuel of the CNS, and research on post-TBI glucose metabolism has typically focused on its energy producing function, as occurs in response to neuronal activation. Due to the sequential processing of glucose through anaerobic glycolysis and subsequent oxidation, increased lactate after TBI was generally seen as evidence of inadequate oxygen delivery or reduced cerebral blood flow. Profound post-traumatic hyperglycemia, which increases damage via lactic acidosis in association with cerebral ischemia, leads to poor outcome, and recent critical care trends towards tight control of blood glucose in all ICU patients. However, some of these "basic principles" of patient care and post-TBI cerebral metabolism have recently come under scrutiny and intense research. Despite normal or elevated levels of blood glucose, interstitial glucose concentrations are often low in TBI patients – this can occur without secondary events (e.g. seizures or ischemia,) and is associated with poor outcomes. Importantly, use of the recommended tight glycemic control can decrease interstitial glucose, increase markers of cellular distress, and increase the oxygen extraction fraction. It also seems that a substantial percentage of glucose is shunted to the pentose phosphate pathway after TBI, potentially used for antioxidant defence, while oxidative pathway flux is reduced. Since discovery

of the "astrocyte-neuronal shuttle" many studies indicate that lactate is a key metabolic substrate supporting neuronal functions. Patients demonstrate cerebral uptake of lactate in the first days after TBI, and administration of lactate (or pyruvate) has demonstrated therapeutic efficacy in animal models of TBI. Administration of ketone bodies has also shown therapeutic efficacy, particularly after TBI in juvenile models. These studies suggest that the rules or principles applicable to cerebral metabolism in the normal CNS have been fundamentally changed after TBI. Further studies on the issues of fuel preference in the traumatically injured brain and the uses of metabolic substrates in the post-TBI pathophysiological environment are urgently needed, given their potential to impact on current clinical management and studies designed to improve cellular survival and recovery of function.

Supported by the UCLA Brain Injury Research Center

Objectives:

- 1) Review the metabolic cascade following traumatic brain injury
- 2) Reveal the changes in the use of basic fuels and of oxygen
- 3) Review the concept of metabolic therapy
- 4) Address the secondary injury issue of cerebral ischemia and hyperglycemia.

L16 WINTR

TRANSLATIONAL RESEARCH GRANTS IN NEUROTRAUMA

Ramona Hicks, NIH/NINDS

Recent discoveries across a broad range of research areas in the neurosciences, including neurotrauma, offer promising opportunities for therapy development. As part of its mission to reduce the burden of neurological disease, the National Institute of Neurological Disorders and Stroke (NINDS) has created several grant mechanisms to facilitate the "translation" of these discoveries into new treatments. However, over the past 5 years, few proposals for translational research for neurotrauma have been successful at obtaining funding. The goal of this presentation is to describe both the opportunities and the challenges associated with the NINDS translational research grant mechanisms. Strategies for overcoming the challenges will also be discussed.

Objectives:

- 1) Understand the rationale for promoting translational research at NIH/NINDS.
- 2) Be aware of the multiple funding mechanisms available at NIH/NINDS for translational research.
- 3) Understand the challenges associated with research on this topic.
- 4) Be informed of strategies for submitting a successful grant application for neurotrauma translational research.

L17

ROLE OF PEROXYNITRATE IN SECONDARY SPINAL CORD INJURY

Linda Noble-Haeusslein, UCSF

Reactive species (RS, including superoxide [$O_2^{\bullet-}$], hydrogen peroxide, hydroxyl radicals, nitric oxide [$^{\bullet}NO$], peroxynitrite [$ONOO^{\bullet}$] etc), produced by acute mechanical injury can cause secondary damage to cells after spinal cord injury (SCI). $ONOO^{\bullet}$, formed by $^{\bullet}NO + O_2^{\bullet-}$, has strong oxidizing and nitrating properties and can damage tissue. Here we report our investigations of the role of $ONOO^{\bullet}$ in SCI. 1) The time courses of RS formation and oxidative product generation were measured. We demonstrated that $O_2^{\bullet-}$, $^{\bullet}NO$, $ONOO^{\bullet}$ and the products of membrane lipid peroxidation (MLP), oxidation of proteins and DNA, and protein nitration were all significantly elevated following SCI. 2) Then $ONOO^{\bullet}$ was generated in an uninjured rat spinal cord at the level produced by SCI and the damage it caused was characterized. We demonstrated that the SCI-produced level of $ONOO^{\bullet}$ is sufficient to cause oxidation and

nitration of proteins, DNA oxidation, MLP, necrotic and apoptotic cell death, and neurological deficits. This direct in vivo evidence demonstrates that $ONOO^{\bullet}$ is an important secondary damage agent. 3) Mn (III) tetrakis (benzoic acid) porphyrin (MnTBAP) - a cell-permeable superoxide dismutase mimetic and a broad spectrum reactive species scavenger - was then administered to test whether oxidative damage and cell loss can be attenuated by this scavenger. Our results show that MnTBAP + nitro-L-arginine (an inhibitor of $^{\bullet}NO$ synthase) significantly reduced the numbers of nitrotyrosine (a marker of protein nitration)-positive cells in both gray and white matter, prevented neurofilament protein from degradation and nerve fiber loss after SCI. MnTBAP alone significantly reduced $ONOO^{\bullet}$ -induced MLP, oxidation and nitration of proteins. Post-SCI treatment with MnTBAP attenuated protein nitration in neurons, astrocytes, and oligodendrocytes. These results further support that $ONOO^{\bullet}$ plays an important role in secondary SCI by causing oxidative damage to the major cellular components, and that its destructive effect can be attenuated by antioxidant therapy with MnTBAP.

Objectives:

L18

OXIDATIVE LIPIDOMICS IN TRAUMATIC BRAIN INJURY

Hulya Bayir, Univ. of Pittsburgh

The role of protein post-translational modifications and protein-protein interactions in different signalling pathways has been comprehensively investigated using the advances in proteomic technology. The role of lipid modifications as well as lipid-protein interactions in the pathophysiology of disease states, however, has been understudied due to the lack of analytical techniques sensitive enough for lipidome analysis. Oxidative lipidomics is a new and exciting research field studying signalling mechanisms by oxidatively modified lipids. It combines biochemical and biophysical approaches with concepts and techniques of free radical chemistry and cell physiology. Using oxidative lipidomics we have shown that a mitochondria specific phospholipid, cardiolipin, is selectively oxidized in neurons in culture after an apoptotic stimulus and in cerebral cortex after experimental traumatic brain injury. By applying electrospray ionization mass spectrometry, we were able to identify molecular species of cardiolipin undergoing peroxidative attack and characterize individual cardiolipin peroxidation products. Furthermore, altering fatty acids in cardiolipin to a more oxidation vulnerable composition rendered cells prone to apoptosis. Use of oxidative lipidomics for mechanistic understanding of lipid-protein interactions and signalling mechanisms by oxidatively modified lipids may have significant translational potential for development of therapies for traumatic brain injury. Support: AHA 0535365N, HD05758.

Objectives:

L19

IS INTERRUPTION OF OXIDATIVE DAMAGE ALONE ENOUGH TO ACHIEVE CLINICALLY MEANINGFUL NEUROPROTECTION?

Edward D. Hall, University of Kentucky Spinal Cord & Brain Injury Research Center

Recent work in our laboratory has demonstrated that generation of the potent reactive oxygen species (ROS) peroxynitrite (PN) is responsible much of the secondary oxidative damage by lipid peroxidation (LP) and protein modification (carbonylation and tyrosine nitration) to mitochondrial and other cellular elements that occurs during the first post-traumatic hrs and days after either diffuse or focal traumatic brain injury (TBI) or contusion spinal cord injury (SCI). In both TBI and SCI, the PN-mediated oxidative damage leads to mitochondrial dysfunction and ultimately failure due to mitochondrial permeability transition (MPT). As a consequence of oxidative compromise of mitochondrial function including calcium (Ca^{++}) buffering), posttraumatic intracellular Ca^{++} overload is exacerbated leading

to calpain-mediated cytoskeletal degradation, neurodegeneration and neurological impairment. We have further shown that early treatment with the nitroxide antioxidant tempol, a potent catalytic scavenger of PN-derived free radicals, can attenuate posttraumatic brain oxidative damage and preserve mitochondrial function and partially reduce cytoskeletal damage and neurodegeneration. However, our results show that the therapeutic window for antioxidant neuroprotection is limited to the first one to two hrs, and that even with very early treatment, the neuroprotective effect is only partial. Accordingly, we have begun to explore whether a neuroprotective strategy that combines a PN radical scavenger such as tempol with either an inhibitor of LP, MPT or calpain might be able to achieve a greater degree of neuroprotection than that produced by scavenging of PN generated radicals or any of the other secondary injury mechanisms alone. Thus, our ongoing experiments are directed at exploring the efficacy and therapeutic window associated with an interruption of the post-traumatic secondary injury cascade at multiple points by combining neuroprotective doses of a PN-directed antioxidant with either previously demonstrated neuroprotective doses of the LP inhibitor U-83836E, the calpain inhibitors MDL28170 or A-705253 or the MPT inhibitor cyclosporine A. This combination approach may produce a quantitatively greater neuroprotective effect that may also prolong the therapeutic window for inhibition of secondary damage after TBI and SCI which should have a greater chance of translational success in future clinical trials. (Supported by 1R01 NS046566; 1P01 NS058484; 1P30 NS051220 and the Kentucky Spinal Cord & Head Injury Research Trust)

Objectives:

- 1) Learn about the spatial and temporal profile of reactive oxygen-induced oxidative damage after acute traumatic brain injury (TBI) and spinal cord injury (SCI);
- 2) Understand the relationship between oxidative damage and other secondary injury events, including mitochondrial dysfunction and the calcium-overload-induced, calpain-mediated proteolytic damage, after TBI or SCI;
- 3) Be familiar with the neuroprotective effects of individual neuroprotective strategies in models of acute TBI and SCI that target either oxidative damage, mitochondrial failure or calpain-mediated proteolysis;
- 4) Understand the rationale for combination, mechanistically complementary, pharmacological therapies to achieve maximal neuroprotection after acute TBI or SCI.

L20 SURGICAL DECOMPRESSION OF CEREBRAL CONTUSIONS

Takeshi Maeda, Tokyo University

Early massive edema caused by severe cerebral contusion results in progressive intracranial pressure elevation and clinical deterioration within 24-72 hours post-trauma. Our clinical studies, including diffusion magnetic resonance imaging, demonstrated that the central (core) area of the contusion undergoes necrosis, whereas the peripheral (rim) area shows cellular swelling caused by ischemia during this period. We suggest that the early massive edema is attributable to a high osmolality within the necrotic brain tissue which may generate an osmotic potential across the central and peripheral areas. We analyzed the effects of surgical excision of the necrotic brain tissue in patients with cerebral contusion registered on the Japan Neurotrauma Data Bank. A group of patients treated conservatively demonstrated a clearly higher mortality at 6 months post-trauma, as compared to a group of patients treated surgically (48% vs. 23%; $p=0.0001$; $n=182$; 1998-2001. 44% vs. 23%; $p=0.004$; $n=175$; 2004-2006). The most striking differences were observed in patients who demonstrated "talk and deteriorate" (56% vs. 17%; $p=0.017$; $n=45$; 1998-2001. 80% vs. 20%; $p=0.00017$; $n=64$; 2004-2006). These findings support our hypothesis that early massive edema is caused by the cerebral contusion through the presence of necrotic brain tissue, and indicate that surgical excision of the

necrotic brain tissue is the only therapy which can avoid clinical deterioration in many cases.

Objectives:

- 1) Learn the mechanism of progressive ICP elevation and clinical deterioration following brain injury.
- 2) Understand the pathophysiology of the early massive edema caused by severe cerebral contusion.
- 3) Describe the relation the early massive edema and a high osmolality within the necrotic brain tissue.
- 4) Evaluate the result of surgical excision of the necrotic brain tissue in patients with cerebral contusion.

L23 DECOMPRESSIVE CRANIECTOMY FOR INTERCRANIAL HYPERTENSION

Bizhan Aarabi, Univ. of Maryland

Over 37 years ago, Dr. Ransohoff proposed a rescue surgical procedure i.e. decompressive craniectomy for patients with uncontrollable intracranial pressures harboring acute subdural hematomas. Exactly five years later Dr. Cooper reported discouraging results from another similar clinical study. This controversy has lingered in the neurosurgical community, despite evidence indicating increased interest in this surgical procedure. We may be years away from answering this question: So far, two attempts in the US have failed to materialize, but two prospective trials have been completed-- one in Australia and one in China. Currently there are two PRCT studies under way: DECRA (Australia) and RESCUEicp (UK). Between 10-20 percent of patients with closed head injury will have intractable intracranial hypertension only responsive to decompressive craniectomy. Close to 50 percent of these patients have diffuse injury and another 50% have associated intracranial hematomas-- >30 ml. Decompressive craniectomy supplies additional volume, improves brain compliance, enhances cerebral perfusion and brain tissue oxygenation, and regulates pressure reactivity. From 2000-2004, 104 patient at the Shock Trauma Center (Baltimore, MD) underwent decompressive craniectomy for blunt head injury. Fifty of 104 patients had diffused injury and an added mass to brain swelling was noted in 54 patients. Decompressive craniectomy was performed from 1-14 days after trauma. Follow up of 102 patients indicated better results when decompressive craniectomy was performed for intractable intracranial hypertension due to malignant brain swelling. In this paper we discuss prognostic variables which play key roles in functional outcome and mortality

Objectives:

- 1) Understand the specific indications of decompressive craniectomy in severe head injury.
- 2) Be able to objectively apply ICP thresholds to execute decompressive craniectomy.
- 3) Be able to better manage intractable intracranial hypertension by medical and surgical means

L24 SURGICAL DECOMPRESSION IN PENETRATING TBI

James Ecklund, Inova Fairfax Hospital

Decompressive craniectomy is a second tier management strategy for increased cranial pressure. The transmission of energy to the brain during a penetrating wound is related to the kinetic energy imparted by the impacting projectile. This energy can be the catalyst for a secondary injury cascade leading to severe cerebral edema that is inadequately treated with standard ICP control measures. Unfortunately, warfare generates a large number of casualties with penetrating head injuries, and the military experience has lead to a number of advances in our understanding and treatment of penetrating brain injury. For example, follow-up studies from Vietnam led to a re-evaluation of the extent of debridement required for optimal management of

PBI. The current experience during the global war on terrorism suggests that an edema pattern can develop during exposure to a blast, often associated with a penetrating wound. This observation has necessitated an extensive utilization of decompressive craniectomy by military surgeons during this conflict. This presentation will describe this experience, and the ongoing follow-up studies designed to better understand the appropriate utilization and timing of this procedure for this patient population.

Objectives:

- 1) Outline how wartime experience has influenced changes in the management of penetrating brain trauma.
- 2) Identify the potential management implications of a blast mechanism in penetrating brain injury
- 3) Identify triage challenges, and indicate optimal timing and utilization of decompressive craniectomy in penetrating brain injury.

L25
MODULATING MICROGLIAL ACTIVITY AFTER BRAIN OR SPINAL CORD TRAUMA

Alan Faden, Georgetown University

Microglial associated inflammatory responses are implicated in acute and chronic neurodegeneration. Using microarrays, we found upregulation of numerous microglial inflammatory factors after spinal cord injury (SCI) or traumatic brain injury (TBI) in rats and mice; increased expression of certain genes and associated proteins persisted for months. Similar gene and protein expression changes are found in activated microglial cultures, and appear to contribute to neuronal cell death *in vitro*. We find that metabotropic glutamate receptor 5 (mGluR5), normally found in neurons and able to inhibit caspase dependent neuronal apoptosis, is highly expressed in microglial cultures after lipopolysaccharide (LPS) stimulation. Pre-treatment with the mGluR5 agonist CHPG or apocynin, an inhibitor of microglial factor p22^{phox}, strongly inhibited LPS-induced microglial activation as reflected by decreased proliferation, nitric oxide production, and TNF α secretion. Co-culture of LPS-activated microglia with neurons causes neurotoxicity; pre-treatment of microglia with CHPG or apocynin prior to LPS blocked neuronal cell death. In parallel studies, CHPG or vehicle were infused intrathecally after moderate impact SCI in rats or controlled cortical impact injury in mice. CHPG treatment markedly reduced microglial associated inflammatory factors, including ED1 and p22^{phox}, and chronic behavioral function was significantly improved. In these same models, apocynin reduced lesion volume at 28 days post-injury. Collectively, these studies indicate that mGluR5 and p22^{phox} represent potential targets for attenuating microglial-related inflammation and associated neurotoxicity.

Objectives:

- 1) To understand the role of microglial related inflammation in secondary injury after brain or spinal cord injury.
- 2) To appreciate the existence of multiple microglial phenotypes, both destructive and protective.
- 3) To recognize the existence of chronic inflammation after CNS trauma.
- 4) To understand the role of NADPH oxidase in secondary injury and the implications for therapy.

L26
OLIGODENDROGLIAL DEGENERATION AND REPLACEMENT STRATEGIES IN SCI

Jeffrey Kocsis, Yale University

Oligodendrocytes can degenerate following SCI leading areas of demyelination which disrupts normal impulse conduction. Transplantation of peripheral myelin-forming cells such as olfactory ensheathing cells (OECs) or Schwann cells can both encourage CNS axonal regeneration and form myelin, and thus may provide a valuable tool for cell-based therapies in spinal cord injury patients. Suspensions of OECs from GFP-expressing rats

were transplanted into one of two model systems in the rat, 1) demyelination of the dorsal funiculus or 2) a dorsal hemisection. In the first model system extensive remyelination with a typical Schwann cell pattern was observed in the lesion zone. In the second lesion (transection), five weeks after transplantation the cells survived within the lesion zone and oriented longitudinally along axons that bridged the transection site. Myelinated axons spanning the lesion were observed in discrete bundles encapsulated by a cellular element. Electron micrographs of spinal cords immunostained with an anti-GFP antibody indicated that a majority of the peripheral-like myelinated axons were derived from donor OECs. Open field locomotor behavior was significantly improved in the OEC transplantation group. The regenerated axons remyelinated by the OECs formed nodes of Ranvier with the appropriate Na channel subtype (Nav1.6). Thus, transplanted OECs derived from adult olfactory bulb can survive and orient longitudinally across a spinal cord transection site and form myelin. These data indicate that transplantation of peripheral myelin-forming cells into the CNS can form functional myelin. Whether this myelination contributes to the observed functional recovery is uncertain. The relative contribution of cellular repair versus trophic support for endogenous recruitment of cells, neuroprotection and synaptic plasticity by OECs will be important for a comprehensive evaluation of the potential therapeutic efficacy of OECs as a cell therapy in spinal cord injury. Moreover, as our understanding of potential trophic influences of OECs is expanded, this could suggest novel pharmacological approaches to the treatment of spinal cord injury.

Objectives:

L27
ROLES AND REGULATION OF REACTIVE ASTROGLIA IN SPINAL CORD INJURY

Michael Sofroniew, UCLA

Glial scar formation is a prominent feature of spinal cord injury (SCI). The functions of reactive, scar forming astrocytes are incompletely understood. Although scar forming reactive astrocytes are often regarded as uniformly detrimental to clinical outcome, both harmful and beneficial activities have been attributed to these cells. Signalling mechanisms that regulate astrocyte reactivity and scar formation after SCI are not well defined. These signalling mechanisms are important because they determine not only the degree to which reactive astrocytes maintain, modify or suppress the functions that astrocytes normally execute in uninjured tissue, but also whether or not reactive astrocytes initiate new, injury-induced activities that may be beneficial or harmful. Our laboratory has been using various kinds of transgenic mouse models to study the activities and regulation of reactive astrocytes after CNS insults *in vivo*. Our studies using transgenically targeted astrocyte ablation point towards roles for reactive astrocytes in restricting inflammation and protecting neurons and oligodendrocytes, thereby helping to limit tissue degeneration and preserve function after CNS injury. Recent findings using the Cre-loxP system for conditional gene deletion in astrocytes indicate that STAT3 signalling is a critical regulator that is required for certain aspects of reactive astrogliosis, including cell hypertrophy, intermediate filament production and glia scar formation, and provide further evidence that scar-forming astrocytes restrict the spread of inflammatory cells after SCI. A better understanding of astrocyte signalling mechanisms may lead to novel strategies to attenuate negative aspects and augment positive aspects of astrocyte reactivity after SCI. (Supported by NIH-NINDS, CA Roman Reed Fund for SCI, Adelson Medical Foundation).

Objectives:

L28
SERUM AND CSF BIOMARKERS IN THE NEURO ICU

Claudia Robertson, Baylor University

Biomarkers of brain injury have been developed from two general strategies. First, protein or protein degradation products of cells have been used as measures of damage to cell structures. These structural markers include S100b, neuron-specific enolase, myelin basic protein, alpha II spectrin, tau, and synaptophysin. Second, mediators of secondary injury or repair have been studied as measures of injury. Cytochrome c, which triggers apoptotic cell death, cytokines which participate in the inflammatory response to injury, nitric oxide, and F2 isoprostane have all showed promise in assessing injury severity. Measurements of CSF levels of such biomarkers have generally more specificity to brain injury than serum markers. However, CSF is not always available, especially in less severely injured patients. Biomarkers could have an important role in assessing prognosis early, in detecting progression of brain injury, and in assessing the effects of therapies.

Objectives:

- 1) To review the current status of potential biomarkers of brain injury
- 2) To assess how biomarkers might be used in the neuro ICU for prognosis, and following treatment of the injury

L29 ADVANCED BIOINFORMATICS IN THE ICU *Geoff Manley, UCSF*

The fundamental purpose of neurocritical care is to protect the injured brain. Neurocritical care is delivered in highly specialized intensive care units and trauma centers that rely on a wide array of instrumentation to monitor and treat the physiological state of the patient. The ability to generate an ever increasing amount of clinical data from physiological sensors, bedside equipment, and laboratory tests has now exceeded the ability to record, process, and integrate this high volume of information for routine patient care. We believe that advances in monitoring must be accompanied by advances in methods of high-frequency, multivariate data analysis that integrate the multiple processes occurring in critically ill patients. We have assembled a powerful multidisciplinary team from academic medicine (UC San Francisco), computer science (UC Berkeley), and industry (Intel's Digital Health Group) to develop a high performance information system and novel computational techniques for critical care medicine. A scalable warehouse for neurocritical care data has been established to facilitate multi-institutional collaboration and knowledge discovery. We describe initial work examining the utility of data-driven methods to identify physiological data patterns for patient classification and outcome. Model-based approaches for physiological data filtering and clinical decision support have also been evaluated. Our multidisciplinary collaboration has demonstrated the feasibility of creating a scalable data warehouse for knowledge discovery. We believe that computational and analytical methods previously used primarily for basic and computer science will fuel the development of a new generation of information systems to improve the diagnosis, treatment and outcome of critically ill and injured patients.

Objectives:

L30 BRAIN OXYGEN MONITORING FOR NEUROTRAUMA OR CONTINUOUS EEG FOR NEUROMONITORING FOLLOWING TBI ICU MONITORING FOR NEUROTRAUMA *Paul Vespa, Univ. California Los Angeles*

No abstract received prior to publication.

Objectives:

L31 MILD TBI-CLINICAL OVERVIEW *Mark Lovell, UPMC*

No abstract received prior to publication.

Objectives:

L32 "MILD TRAUMATIC BRAIN INJURY" IS AN OXYMORON *Douglas H. Smith, Univ. Pennsylvania*

Although mild traumatic brain injury (mTBI) affects over 1 million victims each year in the United States, it is generally ignored as a major health issue. However, this 'mild' form of injury induces persisting neurocognitive dysfunction in many of these patients, exacting an enormous emotional and financial toll on society. Despite the prevalence and impact of mTBI, little is known about potential anatomic or mechanistic substrates that are reflected by the clinical manifestations. Nonetheless, there is growing opinion that a mild form of diffuse axonal injury may play a key role in mTBI pathophysiology. To explore this possibility, we have begun to run parallel studies of mTBI patients compared to a swine model of mTBI induced by head rotational acceleration. At 2-4 days after injury, conventional MRI showed no abnormalities, but advanced neuroimaging techniques elucidated distinct changes throughout the white matter in both human and swine. Histopathological examination of the swine brains demonstrated that the signal changes found with neuroimaging correlated with regions of axonal pathology. In addition, surrogate protein markers of brain pathology were identified after mTBI in the serum of both patients and swine. A potential mechanism of axon dysfunction and degeneration due to mTBI was elucidated using the swine mTBI model and an in vitro model of mild traumatic axonal injury. Specifically, we found that dynamic mechanical deformation of axons induces acute sodium channel (NaCh) dysregulation and a chronic NaCh-opathy. We conclude that mTBI can induce axonal pathology and therefore should not be considered inconsequential. Indeed, the observation that brain pathology can be detected in mTBI patients calls for much more extensive efforts to prevent, diagnose and treat mTBI. Supported by NIH grants, NS38104 and NS056202.

Objectives:

L33 MILD TRAUMATIC BRAIN INJURY AND POST-CONCUSSIVE SYMPTOMS IN MILITARY PERSONNEL *Charles Hoge, Walter Reed Army Institute of Research*

Mild traumatic brain injury (mTBI) has been labeled a "signature" injury of the current wars in Iraq and Afghanistan, based on reports that as many 20% of troops have suffered a mild TBI, often in association with exposure to blast explosions. This has led to population-level screening for mTBI and other efforts by DoD and VA to identify and mitigate the health effects attributed to mTBI. However, because of limited evidence-based studies to guide public health policy, these efforts have been developed largely on the basis of consensus and expert opinion. This talk will examine the interface between evidence and clinical lore for mTBI, drawing on civilian literature as well as recent data collected among returning veterans from Iraq and Afghanistan. Topics that will be discussed include the case definitions and prevalence of mTBI and post-concussive symptoms among returning veterans, the "overlap" between mTBI and PTSD, the assumptions underlying current screening efforts, and recommended best practices for the evaluation and treatment of mTBI and post-concussion symptoms among combat veterans.

Objectives:

L34 THE NIH EFFORT TO DEVELOP A NEW TBI CLASSIFICATION TO FACILITATE THERAPEUTIC TRANSLATION *Geoff Manley, UCSF*

The heterogeneity of traumatic brain injury (TBI) is considered one of the most significant barriers to finding effective therapeutic interventions. In October, 2007, the National Institute of Neurological Disorders and Stroke convened a workshop to outline the steps needed to develop a reliable, efficient and valid classification system for TBI that could be used to link specific patterns of brain and neurovascular injury with appropriate therapeutic interventions. Currently, the Glasgow Coma Scale (GCS) is the primary selection criterion for inclusion in most TBI clinical trials. While the GCS is extremely useful in the clinical management and prognosis of TBI, it does not provide specific information about the pathophysiologic mechanisms which are responsible for neurological deficits and targeted by interventions. On the premise that brain injuries with similar pathoanatomic features are likely to share common pathophysiologic mechanisms, participants proposed that a new, multidimensional classification system should be developed for TBI clinical trials. It was agreed that preclinical models were vital in establishing pathophysiologic mechanisms relevant to specific pathoanatomic types of TBI and verifying that a given therapeutic approach improves outcome in these targeted TBI types. In a clinical trial, patients with the targeted pathoanatomic injury type would be selected using an initial diagnostic entry criterion, including their severity of injury. Coexisting brain injury types would be identified and multivariate prognostic modeling used for refinement of inclusion/exclusion criteria and patient stratification. Outcome assessment would utilize endpoints relevant to the targeted injury type. Advantages and disadvantages of currently available diagnostic, monitoring, and assessment tools were discussed. Recommendations will be presented for enhancing the utility of available or emerging tools in order to facilitate implementation of a pathoanatomic classification approach for clinical trials.

Objectives:

- 1) Aware of an NIH workshop and subsequent funding opportunity for research on Combination Therapies for TBI.
- 2) Understand the rationale for promoting research on this topic.
- 3) Understand the challenges associated with research on this topic.
- 4) Informed of the major recommendations put forward at the workshop for facilitating and optimizing research in this area.

L35

COMBINATION THERAPIES FOR TBI

Ramona Hicks, NIH/NINDS

Traumatic brain injury (TBI) is a major medical problem for which there are no proven interventions. Neuroprotection remains a high priority for TBI, but all Phase III clinical trials to date have failed. A major reason cited for the failures is that the evolving, constellation of pathophysiological mechanisms associated with TBI cannot be addressed with a single "magic bullet". Despite this realization, and the dramatic gains that have resulted from the use of combination therapies for other medical disorders, almost all preclinical and clinical studies for TBI continue to evaluate single interventions. In order to facilitate research on combination therapies for TBI, the NIH convened a workshop in February, 2008. The goal was to develop a rational strategy for selecting and testing combination therapies for TBI. Specifically, workshop participants were asked to: 1) identify the issues and challenges of testing combination therapies in clinical and pre-clinical studies; and 2) propose research methodologies and study designs to overcome these issues; and 3) describe potential synergistic and antagonistic effects of combining some of the currently most promising mono-therapies. Break out groups discussed and recommended strategies for testing combination therapies using in vitro and in vivo models, as well as clinical trials. Major recommendations included: 1) developing an in vitro high throughput screening platform for the evaluation of mono- and multi-drug therapies; 2) developing clinically relevant guidelines for standard of care and outcome measures for pre-clinical studies; 3) selecting combination therapies based on their potential for additive effects when administered either concurrently or sequentially; and 4) using therapies that target multiple mechanisms to provide effective treatment for the broad spectrum of pathophysiological consequences that often occur following TBI.

Objectives:

ABSTRACT POSTER PRESENTATIONS

*Sorted By
Poster Session & Number*

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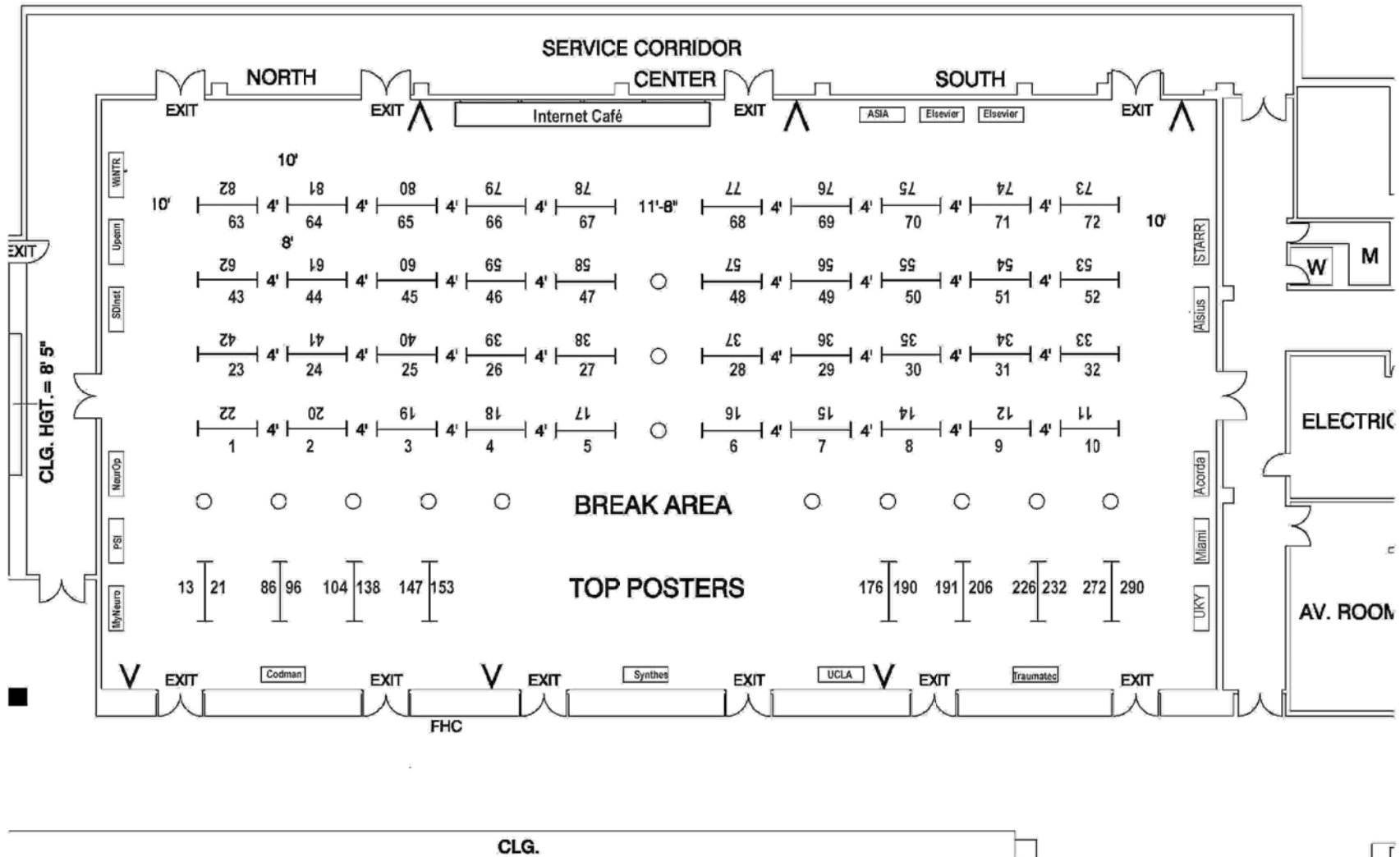
TOP 16 STUDENT POSTERS

P#	TITLE	ASPECTS STUDIED	AREA	First	Last	INSTITUTIONS	Authors
13	ALTERED SERUM 8-ISOPROSTANE LEVELS AFTER TRAUMATIC BRAIN INJURY IN RATS AND HUMANS	Cellular-Molecular Signaling Pathways	TBI	Hehong	Chen	University of Pittsburgh, Tianjin Huanhu Hospital	<u>Chen Hehong</u> , Yan Hong, Ma Xiecheng, Dixon Edward
21	FUNCTIONAL REGENERATION OF SENSORY AXONS INTO TOPOGRAPHICALLY CORRECT AREAS OF THE SPINAL CORD WITH NEUROTROPHIN TREATMENT IN A MODEL OF BRACHIAL PLEXUS INJURY	Electrophysiology, Anatomy/Histology/Imaging	SCI	Pamela	Harvey	Tufts University School of Medicine	<u>Harvey Pamela</u> , Cariani Peter, Frank Eric
86	THREE-DIMENSIONAL CORTICAL CHANGES IN TRAUMATIC AXONAL INJURY	Anatomy/Histology/Imaging	TBI	Teddy	Youn	University of Texas, Brown University	<u>Youn Teddy</u> , Wang Jun, Marquez De La Plata Carlos, George Anuh, Jeon Tina, Ding Kan, Moore Carol, Harper Caryn R, Mumphy Marysa, Devous Sr. Michael, King Richard, Madden Christopher, Diaz-Arrastia Ramon
96	A LINK BETWEEN A NEPRILYSIN GENE POLYMORPHISM AND THE DEVELOPMENT OF AMYLOID- β PLAQUES FOLLOWING TRAUMATIC BRAIN INJURY IN HUMANS	Cellular-Molecular Signaling Pathways, Anatomy/Histology/Imaging	TBI	Victoria E.	Johnson	Univ. of Pennsylvania, Southern General Hospital NHS Trust	<u>Johnson Victoria E.</u> , Stewart William, Graham David I., Stewart Janice E., Praetgaard Amy H., Smith Douglas H.
104	GDNF modifies astroglial responses at graft-host interfaces allowing robust axonal regeneration into Schwann cell-seeded guidance channels grafted into hemisectioned adult rat spinal cords.	Growth Factors/Regeneration/Plasticity	SCI	Ling-Xiao	Deng	Univ. of Kentucky, Indiana University School of Medicine	<u>Deng Ling-Xiao</u> , Hu Jianguo, Liu Nai-Kui, Smith George M., Xu Xiao-Ming
138	OUTCOME IN BLUNT HEAD TRAUMA PATIENTS WITH GLASGOW COMA SCORE OF THREE ON PRESENTATION	Therapeutic Intervention	TBI	Roukoz	Chamoun	Baylor College of Medicine	<u>Chamoun Roukoz</u> , Robertson Claudia, Gopinath Shankar
147	Treatment of trauma-induced AMPA receptor modification by perturbing PDZ protein:protein binding	Therapeutic Intervention, Cellular-Molecular Signaling Pathways	TBI	Joshua	Bell	Univ. of Toronto, St. Michael's Hospital	<u>Bell Joshua</u> , Park Eugene, Baker Andrew
153	THE IY-EGFP-KI MOUSE MODEL REVEALS PREVIOUSLY UNIDENTIFIED POPULATIONS OF HEMATOGENOUS AND MICROGLIAL MACROPHAGES FOLLOWING SPINAL CORD INJURY	Therapeutic Intervention, Inflammation/Neurotoxicity	SCI	Sakina	Thawer	University of Western Ontario	<u>Thawer Sakina</u> , Dekaban Gregory
176	MECHANISMS OF THE EFFECT OF HYPERTONIC SALINE VERSUS MANNITOL ON LEUKOCYTE ADHERENCE AND CELL SURVIVAL AFTER TRAUMATIC BRAIN INJURY IN VITRO	Cellular-Molecular Signaling Pathways	TBI	Donna	Watson	University of Miami School of Medicine	<u>Watson Donna</u> , Huang Tingting, He Dansha, Kuluz John
190	INFLAMMATORY ACTIVATION IS REQUIRED FOR NT-3-INDUCED NEUROPLASTICITY IN INJURED SPINAL CORD	Growth Factors/Regeneration/Plasticity, Inflammation/Neurotoxicity	SCI	Qin	Chen	Baylor College of Medicine, Univ. of Kentucky	<u>Chen Qin</u> , Smith George, Shine H. David
191	BEHAVIOURAL OUTCOME FOLLOWING TRAUMATIC BRAIN INJURY AND SOLUBLE NOGO-66 RECEPTOR THERAPY IN MICE	Growth Factors/Regeneration/Plasticity, Model Characterization/Behavioral Measures	TBI	Anders	Hanell	Uppsala University, Yale University, Biogen Inc.	<u>Hanell Anders</u> , Clausen Fredrik, Björk Maria, Jansson Kristine, Weinreb Paul, Hillered Lars, Strittmatter Stephen, Marklund Niklas
206	DEVELOPMENT OF A MODEL OF PRIMARY BLAST BRAIN INJURY: SHOCKWAVES AS A DIRECT CAUSE OF BRAIN INJURY.	Model Characterization/Behavioral Measures	TBI	Eugene	Park	Univ. of Toronto, St. Michael's Hospital	<u>Park Eugene</u> , Bell Joshua, Liu Elaine, Gottlieb James, Baker Andrew
226	TEMPORAL PROFILE OF MITOCHONDRIAL SUPEROXIDE GENERATION AND APOPTOSIS AFTER MECHANICAL STRETCH IN CORTICAL NEURONS	Therapeutic Intervention, Cellular-Molecular Signaling Pathways	In Vitro Injury	Minke	Tang	Univ. of Pittsburgh, Univ. of Pennsylvania	<u>Tang Minke</u> , Sarnaik Ajit, Feng Weihong, Clark Robert, Kochanek Patrick, Kagan Valerian, Meaney David, Bayr Hülya
232	RESTORATION OF IMPAIRED EXPERIENCE-DEPENDENT PLASTICITY BY ENHANCING NMDA RECEPTOR MEDIATED SYNAPTIC TRANSMISSION AFTER DEVELOPMENTAL TBI	Therapeutic Intervention, Growth Factors/Regeneration/Plasticity	TBI	Naomi	Santa Maria	UCLA	<u>Santa Maria Naomi</u> , Baquing Mary Anne, Hovda David, Giza Christopher
272	PARP-1 POLYMORPHISMS IN HUMANS AFTER SEVERE TRAUMATIC BRAIN INJURY	Growth Factors/Regeneration/Plasticity, Inflammation/Neurotoxicity	TBI	Ajit	Sarnaik	University of Pittsburgh	<u>Sarnaik Ajit</u> , Fink Ericka, Conley Yvette, Okonkwo David, Kochanek Patrick, Clark Robert
290	LONGITUDINAL STUDIES OF SPINAL CORD CONTUSION INJURY USING MAGNETIC RESONANCE IMAGING	Model Characterization/Behavioral Measures, Anatomy/Histology/Imaging	SCI	Laura	Sundberg	University of Texas Medical School	<u>Sundberg Laura</u> , Herrera Juan, Narayana Ponnada

TLC - NATIONAL NEUROTRAUMA SYMPOSIUM

JULY 27 - 29, 2008
HILTON IN THE WALT DISNEY WORLD RESORT
LAKE BUENA VISTA, FLORIDA

INTERNATIONAL BALLROOM



P#	TITLE	ASPECTS	AREA	FIRST	LAST	INSTITUTION	ALL AUTHORS
1	CLINICAL VALUE OF TRACTOGRAPHY IN THE SPINAL CORD	Anatomy/Histology/Imaging	SCI	Eve	Tsai	University of Ottawa, Ottawa Health Research Institute	Tsai Eve, Alkherayf Fahad, Olariu Elena, Berthiaume Alain, Nguyen Thanh Binh, Santos Marlise P. Don, Benoit Brien G., Cameron Ian
2	LONGITUDINAL MRI INVESTIGATION OF NEURONAL AND VASCULAR CHANGES IN SYRINGOMYELIA INDUCED BY CONTUSIVE TRAUMA	Anatomy/Histology/Imaging	SCI	Mehmet	Bilgen	Medical University of South Carolina	Bilgen Mehmet
3	DETECTION OF (PATHOLOGICAL) NEUROLOGICAL CHANGES (IN WHITE MATTER) AND EVALUATION OF PROGNOSIS BY DIFFUSION TENSOR IMAGES	Anatomy/Histology/Imaging	TBI	Makoto	Matsushita		Matsushita Makoto, Hosoda Kohkichi, Hara Yoshie, Yamashita Haruo, Naitoh Yasuo, Kohmura Eiji
4	A PEDIATRIC COMPUTED TOMOGRAPHY SCAN CLASSIFICATION SYSTEM IN CHILDREN WITH SEVERE TRAUMATIC BRAIN INJURY	Anatomy/Histology/Imaging	TBI	Ashley	Di Battista	University of Toronto	Zelsman Melissa, Di Battista Ashley, Brady Ken, Natale Joanne, Shaffner Donald, Guerguerian Anne-Marie
5	MOSSY FIBER PATHWAY ALTERATIONS FOLLOWING EXPERIMENTAL TBI IN THE IMMATURE, POST NATAL DAY (PND) 7 AND 17, RAT	Anatomy/Histology/Imaging	TBI	Frances	Zhang	University of Pittsburgh	Zhang Frances, Card Patrick, Vanino Dana, Adelson P. David
6	ANALYSIS OF THE HIPPOCAMPUS INHIBITORY POPULATION AFTER TRAUMATIC BRAIN INJURY OF GAD67-GFP MICE	Anatomy/Histology/Imaging	TBI	Edgardo	Arroyo	University of Pennsylvania	Arroyo Edgardo J., Elkind Jaclynn, Nyengaard Jens Randel, Witgen Brent, Schwarzbach Elizabeth, Xiong Guoxiang, Cohen Akiva S., Grady M. Sean
7	DEVELOPMENTAL CHANGES IN REGIONAL MECHANICAL PROPERTIES OF THE RAT HIPPOCAMPUS AND CORTEX	Anatomy/Histology/Imaging	TBI	Ben	Elkin	Columbia University	Elkin Benjamin S., Azeloglu Evren U., Costa Kevin D., Morrison III Barclay
8	EFFECT OF PATHOLOGIC EXTRASYNAPTIC NMDA RECEPTOR ACTIVATION ON AMPA RECEPTOR PHOSPHORYLATION	Cellular-Molecular Signaling Pathways	In Vitro Injury	Jennifer	Spaethling	University of Pennsylvania	Spaethling Jennifer, Meaney David
9	15-DEOXY-DELTA 12, 14-PROSTAGLANDIN J2 ATTENUATES ENDOTHELIAL-MONOCYTE INTERACTION: IMPLICATION FOR INFLAMMATORY DISEASES	Cellular-Molecular Signaling Pathways	In Vitro Injury	Mushfiquddin	Khan	Medical University of South Carolina	Giri Shailendra, Prasad Ratna, Khan Mushfiquddin, Singh Avtar, Singh Inderjit
10	SPINALCORDLINK: A NEW WEB-BASED VISUAL INTERFACE FOR ANALYSIS OF A LARGE SPINAL CORD INJURY EXPRESSION PROFILING DATASET	Cellular-Molecular Signaling Pathways	SCI	Susan	Knobloch	The George Washington University School of Medicine	Knobloch Susan, Hoffman Eric, Seo Jinwook
11	APPLICATION OF MULTIPLE LABEL FLUORESCENCE TO VISUALIZE EDEMATOUS CHANGE IN CONDITIONS OF BRAIN SWELLING FOLLOWING CORTICAL CONTUSION INJURY IN RAT.	Cellular-Molecular Signaling Pathways	TBI	Christina R.	Marmarou	Virginia Commonwealth University	Marmarou Christina R., Taya Keitsuke, Liang Xiuyin, Abidi Nageeb, Okuno Kenje, Mqrmarmarou Anthony
12	LATERAL FLUID PERCUSSION RESULTS IN DIFFERENTIAL MODULATION OF CALCINEURIN ACTIVITY	Cellular-Molecular Signaling Pathways	TBI	Severn	Churn	Virginia Commonwealth University	Low Brian, Register David, Kurz Jonathan, Churn Severn
14	ACID SENSING ION CHANNEL 1A CONTRIBUTES TO NEURON DEATH FOLLOWING LATERAL FLUID PERCUSSION INJURY.	Cellular-Molecular Signaling Pathways, Anatomy/Histology/Imaging	TBI	Gregory	Albert	University of Iowa Hospitals and Clinics	Albert Gregory, Elkind Jaclynn, Howard Matthew, Grady M. Sean, Welsh Michael
15	ALTERED HIPPOCAMPAL BETA-AMYLOID LEVELS IN FIMBRIA-FORNIX LESIONED MICE	Cellular-Molecular Signaling Pathways, Anatomy/Histology/Imaging	TBI	Eric	Abrahamson	University of Pittsburgh	Ikonovic Milos, Isanski Barbara, Abrahamson Eric, DeKosky Steven
16	ELEVATION OF RECEPTOR PROTEIN TYROSINE PHOSPHATASE B AFTER BRAIN INJURY: EVIDENCE FOR REGULATION OF SYNAPTIC PLASTICITY THROUGH POSTSYNAPTIC SUBSTRATES	Cellular-Molecular Signaling Pathways, Growth Factors/Regeneration/Plasticity	TBI	Janna	Harris	Virginia Commonwealth University	Harris Janna, Harris Lesley, Lee Nancy, Phillips Linda
17	REGULATION OF CYCLIC-AMP DEPENDENT PHOSPHODIESTERASES IN TNF-ALPHA AND CNS INJURY-ACTIVATED MICROGLIA	Cellular-Molecular Signaling Pathways, Inflammation/Neurotoxicity	In Vitro Injury	Mousumi	Ghosh	University of Miami Leonard M. Miller School of Medicine	Ghosh Mousumi, Atkins Coleen, Dietric Dalton, Pearse Damien
18	ENHANCED MATRIX METALLOPROTEINASES EXPRESSION IS ASSOCIATED WITH GREATER BLOOD BRAIN BARRIER DISRUPTION FOLLOWING TRAUMATIC BRAIN INJURY IN AGED MICE	Cellular-Molecular Signaling Pathways, Inflammation/Neurotoxicity	TBI	Rajat	Sandhir	KU Medical Center	Sandhir Rajat, Hamilton Eric, Onyszczuk Gregory, Berman Nancy
19	EFFECT OF SECONDARY INSULTS UPON AQUAPORIN-4 WATER CHANNELS AND BLOOD BRAIN BARRIER FOLLOWING EXPERIMENTAL CORTICAL CONTUSION IN RATS	Cellular-Molecular Signaling Pathways, Model Characterization/Behavioral Measures	Ischemic-Hypoxic Injury	Christina R.	Marmarou	Virginia Commonwealth University	Taya Keisuke, Marmarou Christina, Okuno Kenji, Marmarou Anthony

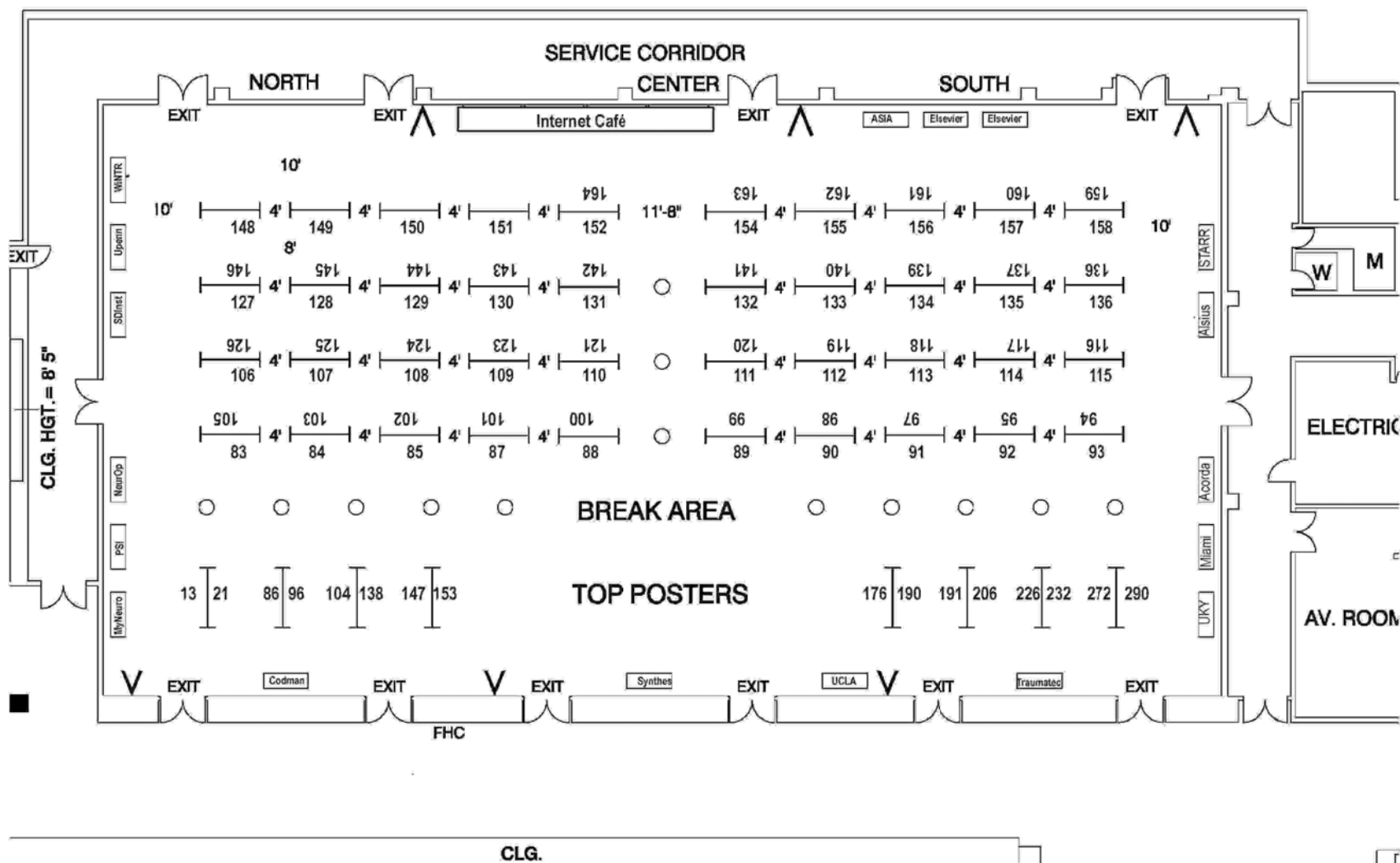
20	AQUAPORIN-4 DELETION REDUCES CEREBRAL EDEMA AND INJURY VOLUME FOLLOWING TRAUMATIC BRAIN INJURY IN MICE	Cellular-Molecular Signaling Pathways, Model Characterization/ Behavioral Measures	TBI	Kazu	Uchida	UCSF	Uchida Kazu, Zador Zsolt, Yao Xiaoming, Verkman Alan, Manley Geoffrey
22	SEVERE SPINAL CORD INJURY DRAMATICALLY REDUCES THE EFFICACY OF PSEUDORABIES VIRUS LABELING OF SYMPATHETIC PREGANGLIONIC NEURONS	Growth Factors/ Regeneration/Plasticity	SCI	Hanad	Duale	University of Kentucky	Duale Hanad, Hou Shaoping, Derbenev Andrei V., Smith Bret N., Rabchevsky Alexander G.
23	MODERATE TRAUMATIC BRAIN INJURY ENHANCES NEURAL PROGENITOR CELL PROLIFERATION WITHOUT INCREASING NEUROGENESIS IN THE ADULT HIPPOCAMPUS	Growth Factors/ Regeneration/Plasticity	TBI	Xiang	Gao		Gao Xiang, Chen Jinhui
24	DENDRITIC AND SYNAPTIC DEGENERATION IN THE HIPPOCAMPAL DENTATE GYRUS FOLLOWING TRAUMATIC BRAIN INJURY	Growth Factors/ Regeneration/Plasticity	TBI	Jinhui	Chen		Gao Xiang, Chen Jinhui
25	"FALSE" REGIONS OF BRAIN ACTIVATION OBSERVED WITH FUNCTIONAL GLUCOSE AUTORADIOGRAPHY AFTER CORTICAL CONTUSION INJURY: CONTRIBUTION OF UNDERLYING PATHOLOGY.	Growth Factors/ Regeneration/Plasticity	TBI	Neil	Harris	UCLA	Lin Sierra, Sutton Richard, Hovda David, Harris Neil
26	INTRASPINAL SPROUTING OF UNMYELINATED PELVIC AFFERENTS AFTER COMPLETE SPINAL CORD INJURY MEDIATES AUTONOMIC DYSREFLEXIA INDUCED BY VISCERAL PAIN	Growth Factors/ Regeneration/Plasticity, Anatomy/Histology/Imaging	SCI	Shaoping	Hou	Univ of Kentucky	Hou Shaoping, Duale Hanad, Rabchevsky Alexander
27	TRANSCRIPTOMIC SCREENING OF MICROVASCULAR ENDOTHELIAL CELLS IMPLICATES NOVEL MOLECULAR REGULATORS OF VASCULAR DYSFUNCTION FOLLOWING SPINAL CORD INJURY	Growth Factors/ Regeneration/Plasticity, Anatomy/Histology/Imaging	SCI	Melissa	Maddie	Kentucky Spinal Cord Injury Research Center (KSCIRC), University of Louisville School of Medicine	Maddie Melissa, Benton Richard, Mahoney Edward, Hagg Theo, Whittemore Scott
28	THE NEUROTROPHIC PROTEIN S100B ENHANCES HIPPOCAMPAL PROGENITOR CELL PROLIFERATION AND SURVIVAL IN A DOSE-DEPENDENT MANNER	Growth Factors/ Regeneration/ Plasticity, Inflammation/Neurotoxicity	TBI	Andrea	Kleindienst	University Erlangen-Nuremberg/ Klinikum Amberg	Gruenbeck Felicitas, Emtmann Irene, Buslei Rolf, Eck Katarina, Koeferl Katharina, Buchfelder Michael, Kleindienst Andrea
29	DESIGNER PG TECHNOLOGY IMPLICATES CHANGES IN GROWTH CONE WIDTH AS A POTENTIAL PREDICTOR OF CHONDROITIN SULFATE PROTEOGLYCAN-INDUCED INHIBITION IN VITRO.	Growth Factors/ Regeneration/ Plasticity, Model Characterization/ Behavioral Measures	SCI	Diane	Snow	The University of Kentucky	Kobraei Eddie, Calulot Chris, Huynh Tru, Westerfield Bailey, Cole Danielle, Tran Binh, Curinga Gabrielle, Hering Thomas, Snow Diane
30	IN VITRO NEUROTOXICITY RESULTING FROM SELECTED NANOMATERIALS	Inflammation/Neurotoxicity	In Vitro Injury	Stephen	Larner	Banyan Biomarkers, Inc.	Larner Stephen F., O'Donoghue Meghan B., Wang Jonathan, Goodman Jared, Wang Kevin K.W.
31	TIME COURSE OF CHANGES IN NADPH OXIDASE FOLLOWING TRAUMATIC BRAIN INJURY	Inflammation/Neurotoxicity	TBI	Stephen	Scheff	University of Kentucky	Ansari Mubeen, Roberts Kelly, Scheff Stephen
32	NEUROPROTECTIVE EFFECTS OF THE PPAR γ AGONIST, ROSIGLITAZONE, AFTER FLUID PERCUSSION BRAIN INJURY	Inflammation/Neurotoxicity	TBI	Debbie	Boone	University of Texas Medical Branch	Boone Debbie, Capra Bridget, Cowart Jeremy, Crookshanks Jeanna, Rojo Daniel, Sell Stacy, Prough Donald, DeWitt Douglas, Hellmich Helen
33	THE SPATIAL AND TEMPORAL PROGRESSION OF SECONDARY BRAIN INJURY AFTER EXPERIMENTAL INTRACEREBRAL HEMORRHAGE	Inflammation/Neurotoxicity, Anatomy/Histology/Imaging	TBI	Jason	Wasserman	Toronto Western Hospital	Wasserman Jason, Schlichter Lyanne
34	MONITORING OF CEREBRAL METABOLISM: NON-ISCHEMIC IMPAIRMENT OF OXIDATIVE AND GLUCOSE METABOLISM FOLLOWING SEVERE TRAUMATIC BRAIN INJURY.	Metabolism/Physiological Assessments	TBI	Jean	Soustiel	Faculty of Medicine, Technion Israel Institute of Technology	Soustiel Jean Francois, Shik Venyamin, Sviril Gil, Zaaroor Menashe
35	DIFFERENTIAL PATTERNS OF RELEASE OF UCHL-1 AND PNFH INTO SERUM AFTER SEVERE TRAUMATIC BRAIN INJURY	Metabolism/Physiological Assessments	TBI	Brian	Blyth	University of Rochester	Blyth Brian, Bazarian Jeffrey, Shaw Gerry
36	BONE LOSS FOLLOWING SPINAL CORD INJURIES OF DIFFERENT SEVERITY IN A RAT MODEL.	Metabolism/Physiological Assessments, Anatomy/Histology/Imaging	SCI	David	Magnuson	University of Louisville	Magnuson David, Brown Edward, Xu Qian, Waddell Seid, Burden Robert, Burke Darlene, Voor Michael
37	DISCRIMINATIVE ABILITY OF S100 PROTEIN TO PREDICT INTRACRANIAL LESION AND CLINICAL OUTCOMES AFTER TRAUMATIC BRAIN INJURY	Metabolism/Physiological Assessments, Anatomy/Histology/Imaging	TBI	Sang Do	Shin	Seoul National University Hospital	Shin Sang Do, Kim Yu Jin, Song Kyoung Jun, Suh Gil Joon
38	ATTITUDES TOWARD ELDERLY INDIVIDUALS WITH SCI: A QUESTIONNAIRE-BASED SURVEY OF REGISTERED NURSES AT AN ACUTE CARE SCI UNIT AND A REHABILITATION SCI UNIT IN CANADA	Model Characterization/ Behavioral Measures	SCI	Julio	Furlan	Toronto Western Research Institute	Furlan Julio, Craven B. Catharine, Fehlings Michael

39	INCREASED CSF LEVELS OF MICROTUBULE ASSOCIATED PROTEIN-2 (MAP-2) HUMAN SUBJECTS WITH SEVERE TRAUMATIC BRAIN INJURY (TBI).	Model Characterization/ Behavioral Measures	TBI	Andrea	Gabrielli	Banyan Biomarkers, Inc.	Hayes Ronald L., Gabrielli Andrea, Buki Andras, Robinson Gillian, Robicsek Steven, Tepas Joseph, Pineda Jose, Robertson Claudia, Oli Monika, Akinyi Linnet, Mo Jixiang, Scharf Dancia, Liu Ming-Cheng, Zheng Wenrong, Wang Kevin
40	THE FENCING RESPONSE AS AN INDICATOR OF TRAUMATIC BRAIN INJURY SEVERITY	Model Characterization/ Behavioral Measures	TBI	Jonathan	Lifshitz	University of Kentucky	Lifshitz Jonathan, Hosseini Ario, Lisembee Amanda
41	VALIDATION OF SPATIAL NOVELTY TASK AS A SENSITIVE PARADIGM TO COGNITIVE DEFICITS RESULTING FROM CCI	Model Characterization/ Behavioral Measures	TBI	Julie	Dobos	University of Pittsburgh	Dobos Julie, Darrah Shaun, Mohler Laura, Wagner Amy
42	CEREBROVASCULAR INJURY IN HEAD TRAUMA – SUSCEPTIBILITY OF BRANCH POINTS	Model Characterization/ Behavioral Measures	TBI	Kenneth	Monson	University of California, San Francisco	Monson Kenneth, Barbaro Nicholas, Manley Geoffrey
43	EDEMA AND CAVITY FORMATION FOLLOWING A MODERATED CORTICAL CONTUSION INJURY IN SPRAGUE DAWLEY RATS	Model Characterization/ Behavioral Measures	TBI	Lesley	Gilmer	University of Kentucky	Gilmer Lesley, Roberts Kelly, Scheff Stephen
44	TRAUMATIC BRAIN INJURY (TBI) IN MICE DEFICIENT IN TUMOR NECROSIS FACTOR RECEPTORS AND FAS: EFFECTS ON HISTOPATHOLOGY AND FUNCTIONAL OUTCOME	Model Characterization/ Behavioral Measures	TBI	Zerong	You		You Zerong, yang Jinsheng, Bruce Hyung Hwan, Hwang Seo Kyoung, Guo Shuzheng, Whalen Michael
45	INJURY SEVERITY IS INDEPENDENT ON THE IMPACT SPEED BETWEEN 0.1 AND 0.4 M/S	Model Characterization/ Behavioral Measures, Anatomy/Histology/Imaging	SCI	Tsang-Wei	Tu	Washington University in St. Louis	Kim Joong H, Tu Tsang-Wei, Huang Jeffery Hun-Ping, Song Sheng-Kwei
46	SERIALY PLACED CONTUSIONS OF THE MEDIAL FRONTAL CORTEX ENHANCES RECOVERY OF FUNCTION IN ADULT RATS	Model Characterization/ Behavioral Measures, Anatomy/Histology/Imaging	TBI	Cole	Vonder Haar	University of Portland	Vonder Haar Cole, Friend Danielle, Peruzzaro Sarah, Smith Jeffrey
47	A NEW MODEL FOR HIGH SPATIAL AND TEMPORAL RESOLUTION IN HUMAN ELECTROPHYSIOLOGY: DECOMPRESSIVE CRANIECTOMY	Model Characterization/ Behavioral Measures, Electrophysiology	TBI	Bradley	Voytek	UCSF	Voytek Bradley, Secundo Lavi, Bidet-Caulet Aurelie, Scabini Donatella, Stiver Shirley, Gean Alisa, Manley Geoffrey, Knight Robert
48	THE EFFECTS OF TRAUMATIC BRAIN INJURY ON CONNEXIN MRNA EXPRESSION IN CEREBRAL ARTERIES OF MALE AND FEMALE RATS	Model Characterization/ Behavioral Measures, Inflammation/Neurotoxicity, Metabolism/Physiological Assessments	TBI	Stacy	Sell	University of Texas Medical Branch	Sell Stacy L., Boone Deborah R., Crookshanks Jeanna M., Cowart Jeremy, Prough Donald S., Hellmich Helen L., DeWitt Douglas S.
49	EVALUATION OF GLUTAMATE TRANSPORTER EXPRESSION IN PURE ASTROCYTE AND NEURON/ASTROCYTE CO-CULTURES FOLLOWING SEVERE MECHANICAL STRAIN INJURY.	Model Characterization/ Behavioral Measures, Metabolism/Physiological Assessments	In Vitro Injury	Justin	Beller	University of California, Davis	Beller Justin A, Berman Robert F, Lyeth Bruce G
50	PROTECTION OF ENDOTHELIAL FUNCTION BY S-NITROSOGLUTATHIONE FOLLOWING ACUTE INJURY IN A RAT MODEL OF EXPERIMENTAL STROKE	Therapeutic Intervention	Ischemic-Hypoxic Injury	Mushfiquddin	Khan	Medical University of South Carolina	Khan Mushfiquddin, Hoda Nasrul, Giri Shailendra, Singh Inderjit, Singh Avtar
51	RETROSPECTIVE ANALYSIS OF THE SAFETY AND EFFICACY OF REHABILITATION ALONE COMPARED TO OLFACTORY MUCOSA AUTOGRAFTS (OMA) AND REHABILITATION USING PHYSICAL THERAPY RECORDS FROM PATIENTS WITH SEVERE, CHRONIC SPINAL CORD INJURY	Therapeutic Intervention	SCI	Jean	Peduzzi	Wayne State University School of Medicine	Dabrowski Edward, Peduzzi Jean, Hinderer Steven, Morgan Lindsey, Larson Cathy, Denison Paula, Sheridan Brian, Meythaler Jay, Lima Carlos
52	ELECTRICAL STIMULATION IN MIDBRAIN OR MEDULLA STARTED SOON AFTER MODERATE CONTUSION OF THORACIC SPINAL CORD BOOSTS SENSORIMOTOR AND ANATOMICAL RECOVERY.	Therapeutic Intervention	SCI	Ian	Hentall	University of Miami	Hentall Ian, Fun Elizabeth, Rodriguez Maria Luisa, Burns Scott
53	MAGNESIUM IN A POLYETHYLENE GLYCOL FORMULATION PROVIDES NEUROPROTECTION AFTER ACUTE SPINAL CORD INJURY	Therapeutic Intervention	SCI	Brian	Kwon	University of British Columbia	Kwon Brian, Roy Josee, Lee Jae, Stammers Anthea, Marx Jeff
54	SURVIVAL AND INTEGRATION OF TRANSPLANTED NERVOUS TISSUE CONSTRUCTS IN SPINAL CORD LESIONS USING TRANSGENIC RATS	Therapeutic Intervention	SCI	Kevin	Browne	University of Penn	Browne Kevin, Iwata Akira, Pfister Bryan, Yuen Tracy, Smith Douglas
56	SAFETY OF CYCLOSPORIN A IN SEVERE TRAUMATIC BRAIN INJURY PATIENTS: RESULTS FROM A PROSPECTIVE, RANDOMIZED TRIAL	Therapeutic Intervention	TBI	Gretchen	Brophy	Virginia Commonwealth University	Brophy Gretchen, Mazzeo Anna Teresa, Robles JR, Gilman Charlotte, Hayes Ronald, Bullock Ross

57	SYSTEMATIC REVIEW ON DECOMPRESSIVE CRANIECTOMY IN CHILDREN FOLLOWING SEVERE TRAUMATIC BRAIN INJURY	Therapeutic Intervention	TBI	Jorn	Fierstra	Toronto: The Hospital for Sick Children	Fierstra Jorn, Grewal Sanjeev, Di Battista Ashley, Merat Michele, Guerguerian Anne-Marie
58	EFFECT OF TAURINE WITH MAGNESIUM SULFATE ON RESPIRATORY CHAIN ENZYMES OF MITOCHONDRION IN RATS WITH ACUTE SEVERE TRAUMATIC BRAIN INJURY	Therapeutic Intervention	TBI	Huiling	Huang	Tianjin Neurosurgical Institute	Huang Huiling, Wang Qin, Wang Chen, Cai Ying
59	POLYNITROXYLATED PEGYLATED HEMOGLOBIN SOLUTION FOR THE ACUTE LIMITED FLUID RESUSCITATION OF HEMORRHAGIC SHOCK AFTER TRAUMATIC BRAIN INJURY IN A MOUSE MODEL	Therapeutic Intervention	TBI	David	Shellington	University of Pittsburgh School of Medicine	Shellington David, Exo Jennifer, Vagni Vincent, Ma Li, Feldman Keri, Clark Robert, Bayir Hulya, Dixon C. Edward, Jenkins Larry, Abuchowski Abe, Hsia Carleton, Kochanek Patrick
60	BRAIN ENERGY DEPLETION CAUSED BY A DIFFUSE HEAD INJURY IN RATS IS NOT AMELIORATED BY THE INFUSION OF SODIUM LACTATE	Therapeutic Intervention	TBI	Christina R.	Marmarou	Virginia Commonwealth University	Parieto Ruth, Tavazzi Barbara, Taya Keisuke, Barrios Laura, Marmarou Christina R., Marmarou Anthony
61	THE CEREBROVASCULAR PROTECTIVE EFFECTS OF COMBINATION THERAPY UTILIZING HYPOTHERMIA AND SOD FOLLOWING MODERATE FLUID PERCUSSION BRAIN-INJURY IN RATS	Therapeutic Intervention	TBI	Enoch	Wei		WEI ENOCH, GAO GUOYI, POVLISHOCK JOHN
62	ENVIRONMENTAL ENRICHMENT CONFERS LONG-LASTING COGNITIVE BENEFITS EVEN AFTER A RELATIVELY BRIEF EXPOSURE AFTER EXPERIMENTAL BRAIN TRAUMA	Therapeutic Intervention	TBI	Jeffrey	Cheng		Cheng Jeffrey P., Sozda Christopher N., Olsen Adam S., Hoffman Ann N., Shaw Kaitlyn E., Kline Anthony E.
63	SALMON-DERIVED FIBRIN FOR THE ACUTE TREATMENT OF CORTICAL ABLATION BRAIN INJURIES	Therapeutic Intervention, Anatomy/Histology/Imaging	TBI	Anthony	Choo	University of Pennsylvania	Choo Anthony M., Winer Jessamine P., Winkelstein Beth A., Smith Douglas H., Janmey Paul A., Meaney David F.
64	LITHIUM TREATMENT FOLLOWING LATERAL FLUID PERCUSSION BRAIN INJURY: DETERMINATION OF THE OPTIMAL THERAPEUTIC DOSE AND ANALYSIS OF NEUROPROTECTIVE EFFECTS	Therapeutic Intervention, Anatomy/Histology/Imaging	TBI	Katharine	Eakin-Merenda	Virginia Commonwealth University	Eakin-Merenda Katharine, Kokiko-Cochran Olga, Hamm Robert
65	THE IMMUNOPHILIN LIGAND FK506 ATTENUATES NEUROFILAMENT COMPACTION BUT NOT IMPAIRED AXONAL TRANSPORT FOLLOWING DIFFUSE BRAIN INJURY IN THE IMMATURE RAT	Therapeutic Intervention, Cellular-Molecular Signaling Pathways	TBI	Ann Mae	DiLeonardi	Drexel University College of Medicine	DiLeonardi Ann Mae, Huh Jimmy, Raghupathi Ramesh
66	TOLL LIKE RECEPTOR-4 PROMOTES CEREBRAL EDEMA FOLLOWING TRAUMATIC BRAIN INJURY IN MICE VIA INCREASED AQUAPORIN-4 EXPRESSION	Therapeutic Intervention, Cellular-Molecular Signaling Pathways	TBI	Melissa	Laird	Medical College of Georgia	Laird Melissa, Wakade Chandramohan, Vender John, Dhandapani Krishnan
67	TREATMENT WITH MONO-ADP-RIBOSYLATION INHIBITORS PROTECT AGAINST THE INCREASED SUSCEPTIBILITY SEEN IN CA1 HIPPOCAMPAL NEURONS IN AGED RATS.	Therapeutic Intervention, Electrophysiology	In Vitro Injury	Roi Ann	Wallis	VA Greater Los Angeles Healthcare System	Wallis Roi Ann, Panizzon Kimberly
68	A COMBINATORIAL STRATEGY USING ADULT NEURAL PRECURSOR CELLS, GROWTH FACTORS AND GLIAL SCAR INHIBITION PROMOTES REMYELINATION AND NEUROBEHAVIORAL RECOVERY IN THE CHRONICALLY INJURED SPINAL CORD	Therapeutic Intervention, Growth Factors/Regeneration/Plasticity	SCI	Soheila	Karimi		Karimi Soheila, Eftekharpour Eftekhar, Wang Jian, Schut Desiree, Fehlings Michael G.
69	IDENTIFICATION AND TESTING OF FACTORS INVOLVED IN NEUROTROPHIN-MEDIATED NEUROPROTECTION OF HIPPOCAMPAL NEURONS AFTER TBI.	Therapeutic Intervention, Growth Factors/Regeneration/Plasticity	TBI	Saafan	Malik	University of Pennsylvania, Center for Brain Injury and Repair	Malik Saafan, Hauspurg Alisse, Watson Deborah
70	NEUROPROTECTIVE FUNCTION OF EGF, BFGF AND IGF-1 IN THE INJURED BRAIN ARE ASSOCIATED TO THEIR NEUROGENIC EFFECTS	Therapeutic Intervention, Growth Factors/Regeneration/Plasticity	TBI	Andrew	Rolfe	Virginia Commonwealth Univ.	Rolfe Andrew, Colello Raymond, Gao Weiyi, Levasseur Joseph, Bullock Ross, Sun Dong
71	A NOVEL ASPRIN IMPROVES LOCOMOTOR RECOVERY IN RATS WITH SPINAL CORD INJURY (SCI)	Therapeutic Intervention, Inflammation/Neurotoxicity	SCI	Shiqiang	Tian	The University of Texas Medical School at Houston	Tian Shiqiang, Stehpen Nicholas A., Few Jonathian, Lichtenberger Lenard M.
72	MELATONIN PREVENTED INFLAMMATION, AXONAL DAMAGE, AND NEURONAL DEATH IN SPINAL CORD INJURY IN RATS	Therapeutic Intervention, Inflammation/Neurotoxicity	SCI	Swapan	Ray	University of South Carolina School of Medicine	Samantaray Supriti, Das Arabinda, Matzelle Denise, Reiter Russel, Ray Swapan, Banik Naren
73	POSTTRAUMATIC SEIZURE SUSCEPTIBILITY IS ATTENUATED BY HYPOTHERMIA THERAPY	Therapeutic Intervention, Inflammation/Neurotoxicity	TBI	Coleen	Atkins	University of Miami Miller School of Medicine	Atkins Coleen M., Truettner Jessie S., Lotocki George, Sanchez-Molano Juliana, Alonso Ofelia F., Bramlett Helen M., Dietrich W. Dalton

74	FOCAL MONITORING (MICRODIALYSIS AND BRAIN TISSUE OXYGEN) IN THE CLINICAL MANAGEMENT OF METABOLIC CRISIS IN SEVERELY HEAD-INJURED PATIENTS.	Therapeutic Intervention, Metabolism/Physiological Assessments	TBI	Emilie	Carre	France	Carre Emilie, Boret Henry, Palmier Bruno, Goutorbe Philippe, Montcriol Ambroise, Bourdon Lionel, Risso Jean-Jacques
75	INCIDENCE OF LOW BRAIN TISSUE OXYGEN IN PATIENTS WITH SEVERE TRAUMATIC BRAIN INJURY	Therapeutic Intervention, Metabolism/Physiological Assessments	TBI	Kristine	O'Phelan	The Queen's Medical Center	O'Phelan Kristine, Sasaki Alana, Albano Melanie, Efrid James, Green Deborah, Chang Cherylee, Johnson Katherine, Beniga Juliet
76	ADRENOMEDULLIN HELPS TO PREVENT SEX DEPENDENT REDUCTION IN THE AUTOREGULATORY INDEX DURING HYPOTENSION AFTER PIGLET BRAIN INJURY.	Therapeutic Intervention, Metabolism/Physiological Assessments	TBI	William	Armstead	University of Pennsylvania	Armstead William, Kofke W. Andrew, Vavilala Monica
77	EFFECT OF OXYGEN DELIVERY ON THE METABOLIC DYNAMICS OF GLUCOSE IN THE NORMAL AND LATERAL FLUID PERCUSSION INJURED RAT BRAIN	Therapeutic Intervention, Metabolism/Physiological Assessments	TBI	Marinella	Gugliotta	Virginia Commonwealth Univ.	Gugliotta Marinella, Levasseur Joseph, Sun Dong, Spiess Bruce, Bullock Ross
78	INTERFERON GAMMA TREATMENT DECREASED CSPG EXPRESSION AND RECOVERD HIND-LIMB FUNCTION AFTER SPINAL CORD INJURY IN MICE	Therapeutic Intervention, Model Characterization/ Behavioral Measures	SCI	Takayuki	Fujiyoshi	Chiba university	Fujiyoshi Takayuki, Kubo Takekazu, Chan Carmen, Yamazaki Masashi, Okawa Akihiko, Koda Masao, Hashimoto Masayuki, Mannoji Chikato, Kawabe Junko, Furuya Takeo, Endo Tomonori, Hayashi Koichi, Takahashi Kazuhisa, Yamashita Toshihide
79	CONTINUOUS SHOCK HAS A PROTECTIVE EFFECT AGAINST THE SPINAL LEARNING DEFICIT INDUCED BY INTERMITTENT SHOCK.	Therapeutic Intervention, Model Characterization/ Behavioral Measures	SCI	Denise	Puga	Texas A & M University	Puga Denise, Hook Michelle, Huie John, Hoy Kevin, Grau James
80	THE COLOMBIAN NEUROTRAUMA CONSORTIUM: A PILOT PROJECT FOR TBI REGISTRY IN A LOW-MIDDLE INCOME COUNTRY.	Therapeutic Intervention, Model Characterization/ Behavioral Measures	TBI	Andres	Rubiano Escobar	University of Pittsburgh	Rubiano Escobar Andres M., Sanchez Alvaro I., Puyana Juan C., Fabio Anthony, Adelson P. David
81	COMPARATIVE NEUROPROTECTIVE EFFECTS OF CYCLOSPORIN A AND NIM811, A NON-IMMUNOSUPPRESSIVE CYCLOSPORIN A ANALOG, FOLLOWING TRAUMATIC BRAIN INJURY	Therapeutic Intervention, Model Characterization/ Behavioral Measures	TBI	Lamin Han	Mbye	University of Kentucky	Mbye Lamin H., Singh Indrapal N., Carrico Kimberly M., Saatman Katherine, Hall Edward D.
82	COGNITIVE AND BEHAVIORAL EFFECTS OF DILANTIN ADMINISTRATION AFTER CONTROLLED CORTICAL IMPACT	Therapeutic Intervention, Model Characterization/ Behavioral Measures	TBI	Laura	Mohler	University of Pittsburgh	Mohler Laura, Cummings Erin, Darrah Shaun, Wagner Amy, Chen Xiangbai, Galang Gary, Chuang Jerry

INTERNATIONAL BALLROOM



POSTER SESSION B – MONDAY 2:30 – 3:45

P#	TITLE	ASPECTS	AREA	FIRST	LAST	INSTITUTION	ALL AUTHORS
84	CYCLOPHILIN D KNOCKOUT, EXCITOTOXIC INSULT, AND SPINAL CORD INJURY	Anatomy/Histology/Imaging	SCI	Jordan	Clark	University of Kentucky	Clark Jordan, Naga Kranthi, Geddes James
85	EVALUATION OF ANATOMICAL AND FUNCTIONAL CONNECTIONS FOLLOWING TRAUMATIC BRAIN INJURY IN HUMANS BY DIFFUSION TENSOR IMAGING AND RESTING-STATE FUNCTIONAL CONNECTIVITY	Anatomy/Histology/Imaging	TBI	Christine	Mac Donald	Washington University	Mac Donald Christine, Lee Amy, Naunheim Rosanne, Vaishnavi Sanjeev, Epstein Adrian, Foster Erin, Snyder Abraham, Chleboun Steffany, Shimony Joshua, Raichle Marcus, Brody David
87	CHARACTERIZATION OF CEREBRAL BLOOD FLOW USING ARTERIAL SPIN MR FOLLOWING EXPERIMENTAL TBI IN IMMATURE POSTNATAL DAY (PND 17) RATS	Anatomy/Histology/Imaging	TBI	Wendy	Fellows-Mayle	University of Pittsburgh	Fellows-Mayle Wendy, Foley Lesley, Hitchens Kevin, Ho Chien, Adelson P. David
88	NEURONAL LOSS AND ATROPHY EXTENDS INTO THE SOMATOSENSORY BARREL CORTEX AFTER MIDLINE FLUID PERCUSSION BRAIN INJURY	Anatomy/Histology/Imaging	TBI	Amanda	Lisembee	University of Kentucky	Lisembee Amanda, Lifshitz Jonathan
89	MICROVASCULAR ALTERATIONS AND RECOVERY CORRELATE WITH INCREASED VEGF EXPRESSION FOLLOWING TWO GRADES OF FLUID PERCUSSION TRAUMA.	Anatomy/Histology/Imaging	TBI	Ishita	Siddiq	Toronto : Dalhousie University	Siddiq Ishita, Park Eugene, Baker Andrew J
90	SYSTEMS BIOLOGY ANALYSES OF MOLECULAR NETWORKS IN RHO-ASSOCIATED KINASE MEDIATED NEURITOGENESIS	Cellular-Molecular Signaling Pathways	In Vitro Injury	Zhiqun	Zhang	Banyon	Zhang Zhiqun, Wang Kevin, hayes Ronald
91	MICROTUBULE INSTABILITY DUE TO TRAUMATIC AXONAL INJURY TRIGGERS DEGENERATION	Cellular-Molecular Signaling Pathways	In Vitro Injury	Min	Tang-Schomer	University of Pennsylvania	Tang-Schomer Min, Patel Ankur, Baas Peter, Weingard Matthew, Meaney Dave, Smith Douglas
92	SPINAL CORD INJURY INDUCES UPREGULATION OF BECLIN 1 AND PROMOTES AUTOPHAGIC CELL DEATH	Cellular-Molecular Signaling Pathways	SCI	Haruo	Kanno	Tohoku University School of Medicine	Kanno Haruo, Ozawa Hiroshi, Itoi Eiji
93	TRAUMATIC BRAIN INJURY BIOMARKERS: A NEUROPROTEOMIC-BASED DISCOVERY, SYSTEMS BIOLOGY-BASED SELECTION AND VALIDATION IN ANIMAL STUDIES	Cellular-Molecular Signaling Pathways	TBI	Kevin	Wang	Banyan Biomarkers, Inc.	Wang Kevin KW, Kobeissy Firas H, Liu Ming Cheng, Oli Monika, Robinson Gillian, Dave Jitendra, Tortella Frank C., Robicsek Steven A., Gabrielli Andrea, Robertson Claudia, Hayes Ronald L.
94	MEMBRANE LOCALIZATION OF VOLTAGE GATED SODIUM CHANNELS FOLLOWING CALPAIN MEDIATED PROTEOLYSIS	Cellular-Molecular Signaling Pathways	TBI	Catherine	von Reyn	University of Pennsylvania	von Reyn Catherine R., Meaney David F.
95	INHIBITION OF CALPONIN PHOSPHORYLATION BY ENDOTHELIN RECEPTOR-A ANTAGONISM AMELIORATES HYPOPERFUSION AND IMPROVES BEHAVIORAL OUTCOME FOLLOWING TRAUMATIC BRAIN INJURY.	Cellular-Molecular Signaling Pathways	TBI	Christian	Kreipke		Kreipke Christian, Schafer Patrick, Schafer Steven, Rafols Jose
97	A TEMPORAL STUDY OF POST SYNAPTIC DENSITY PROTEIN 95 EXPRESSION AFTER TRAUMATIC BRAIN INJURY.	Cellular-Molecular Signaling Pathways, Anatomy/Histology/Imaging	TBI	Chandramohan	Wakade	Medical College of Georgia	Wakade Chandramohan, SR Sangeetha, Laird Mellisa, Dhandapani Krishnan, Vender John
98	EVALUATION OF BCL-2 LEVELS IN CEREBROSPINAL FLUID FOLLOWING SEVERE TRAUMATIC BRAIN INJURY	Cellular-Molecular Signaling Pathways, Inflammation/Neurotoxicity	TBI	Brett	Postal	University of Pittsburgh	Postal Brett, Rogers Emily, Chen Xiangbai, Fabio Anthony, Yuan Zhifang, Niyonkuru Christian, Wagner Amy
99	ACTIVATION OF MITOCHONDRIAL UNCOUPLING PROTEINS BY POLYUNSATURATED FATTY ACID REDUCES REACTIVE OXYGEN SPECIES PRODUCTION	Cellular-Molecular Signaling Pathways, Metabolism/Physiological Assessments	In Vitro Injury	Laurie	Davis	University of Kentucky	Davis Laurie, Bosken Jeff, Rho Jong, Sullivan Patrick
100	LEVELS OF UCH-L1 IN HUMAN CSF AND OUTCOME FOLLOWING SEVERE TRAUMATIC BRAIN INJURY	Cellular-Molecular Signaling Pathways, Metabolism/Physiological Assessments	TBI	Linda	Papa	Orlando Regional Medical Center	Papa Linda, Oli Monika, Akinyi Linnet, Liu Ming Cheng, Zheng Wenrong, Tepas Joseph J., Pineda Jose, Robinson Gillian, Robicsek Steve A., Gabrielli Andrea, Brophy Gretchen, Demery Jason, Heaton Shelley, Robertson Claudia, Hayes Ronald L., Wang Kevin K.W.

101	AUTOMATED MULTIVARIATE FEATURES OF BRAIN ELECTRICAL ACTIVITY PREDICTS SEVERITY OF HEAD INJURY IN THE EMERGENCY DEPARTMENT	Electrophysiology	TBI	Rosanne	Naunheim	Washington University	Naunheim Rosanne, English Joy, Treaster Matt, Prichep Leslie
102	ENGINEERING MULTI-FUNCTIONAL THREE-DIMENSIONAL NERVOUS TISSUE CONSTRUCTS	Growth Factors/Regeneration/Plasticity	PNS Injury	Bryan	Pfister	New Jersey Institute of Technology	Siriwardane Mevan, Tolentino Rosa, Pfister Bryan
103	SURVIVAL OF NEURALLY INDUCED MESENCHYMAL STEM CELLS DETERMINES DEGREE OF FUNCTIONAL RECOVERY IN GRAFTED INJURED SPINAL CORD RATS.	Growth Factors/Regeneration/Plasticity	SCI	Arshak	Alexanian	Medical College of Wisconsin	Alexanian Arshak, Stadig Christy, Maiman Dennis
105	SELECTIVE DEATH OF NEWBORN NEURONS IN HIPPOCAMPAL DENTATE GYRUS FOLLOWING MODERATE EXPERIMENTAL TRAUMATIC BRAIN INJURY	Growth Factors/Regeneration/Plasticity	TBI	Jinhui	Chen		Gao Xiang, Den Ying, Cho Wongil, Carrico Kimberly M., Hall Edward D., Chen Jinhui
106	PRENATAL STRESS INCREASES INJURY SIZE AND NEURONAL MIGRATION FOLLOWING TRAUMATIC BRAIN INJURY	Growth Factors/Regeneration/Plasticity	TBI	Lindsay	Ferguson	Michigan State University	Ferguson Lindsay, Shea Anne, Kozlowski Dorothy
107	A CRITICAL WINDOW FOR AXON SPROUTING OPENS DURING A NET REDUCTION IN THE AMOUNT AND STRUCTURAL ARRANGEMENT OF CSPG GROWTH-INHIBITORY PROTEINS AFTER BRAIN TRAUMA.	Growth Factors/Regeneration/Plasticity	TBI	Neil	Harris	UCLA	Harris Neil, Hovda David, Sutton Richard
108	TRANSPLANTED LP ENCOURAGES AND MODULATES TISSUE REPAIR CONDUCTIVE TO AXONAL REGENERATION	Growth Factors/Regeneration/Plasticity, Anatomy/Histology/Imaging	SCI	Shu-xin	Zhang	Spinal Cord Society Research Center	Zhang Shuxin, Sarmiere Patrick, Doolen Suzanne, Huang Fengfa, White Jason, Gates Mary, Holmberg Eric
109	TRANSCRIPTIONAL ACTIVATION OF SPINAL MICROVASCULAR ENDOTHELIAL CELLS BY TGF[BETA]1 OCCURS CONCOMITANT WITH ACUTE NEUROVASCULAR UNIT (NVU) PLASTICITY FOLLOWING FOCAL GRAY MATTER ISCHEMIA-REPERFUSION SPINAL CORD INJURY (SCI)	Growth Factors/Regeneration/Plasticity, Anatomy/Histology/Imaging	SCI	Richard	Benton	Kentucky Spinal Cord Injury Research Center (KSCIRC), University of Louisville School of Medicine	Benton Richard, Maddie Melissa, Whittemore Scott
110	IMPAIRED EXPRESSION OF NEUROPROTECTIVE MOLECULES IN THE HIF-1 ALPHA PATHWAY FOLLOWING TRAUMATIC BRAIN INJURY IN AGED MICE.	Growth Factors/Regeneration/Plasticity, Inflammation/Neurotoxicity	TBI	Joshua	Anderson	University of Kansas	Anderson Joshua, Sandhir Rajat, Berman Nancy
111	COMBINED EFFECT OF LEAD AND CADMIUM ON DIVALENT METAL TRANSPORTER1 (DMT1) EXPRESSION IN CENTRAL NERVOUS SYSTEM OF BUDDING RAT	Inflammation/Neurotoxicity	In Vitro Injury	Xia	Huo	Shantou University Medical College	Huo xia, Gu chengwu, Xu xinjin
112	GLIAL ACTIVATION LINKS TRAUMATIC BRAIN INJURY AND LONG-TERM VULNERABILITY TO EPILEPSY	Inflammation/Neurotoxicity	TBI	Mark	Wainwright	Northwestern University Feinberg School of Medicine	Chrzasczcz MaryAnn, Nair Sangeetha, Patel Fatima, Van Eldik Linda, Watterson D. Martin, Wainwright Mark
113	NEUROPROTECTIVE EFFECTS OF A NOVEL ANTI-INFLAMMATORY DRUG DERIVED FROM AFRAMOMUM MELEGUETA	Inflammation/Neurotoxicity	TBI	Bridget	Capra	University of Texas Medical Branch	Capra Bridget, Boone Debbie, Cowart Jeremy, Sell Stacy, Prough Donald, DeWitt Douglas, Hellmich Helen
114	TEMPORAL AND SPATIAL DYNAMICS OF PEROXYNITRITE-MEDIATED OXIDATIVE DAMAGE AFTER SPINAL CORD CONTUSION INJURY IN RATS	Inflammation/Neurotoxicity, Anatomy/Histology/Imaging	SCI	Kimberly	Carrico	University of Kentucky	Carrico Kimberly, Hall Edward
115	THE CHANGE IN GLASGOW COMA SCALE IN ALCOHOL INTOXICATED HEAD-INJURED PATIENTS	Inflammation/Neurotoxicity, Metabolism/Physiological Assessments	TBI	Hazem	Shahin	Baylor College of Medicine	Shahin Hazem, Gopinath Shankar P., Robertson Claudia S.
116	CEREBRAL BLOOD FLOW VELOCITY MEASUREMENT IN CHILDREN AT RISK FOR HYPOXIC-ISCHEMIC BRAIN INJURY: PRELIMINARY RESULTS	Metabolism/Physiological Assessments	Ischemic-Hypoxic Injury	Michele	Merat	University of Toronto	Merat Michele, Shibata Audrey, Wagner Bendicht P., Hutchison Jamie, Guerguerian Anne-Marie
117	EFFECTS OF HYPERGLYCEMIA ON OUTCOME IN SEVERE TRAUMATIC BRAIN INJURY (STBI) IN CHILDREN	Metabolism/Physiological Assessments	TBI	Rebecca	Smith	University of Pittsburgh Children's Hospital of Pittsburgh	Smith Rebecca, Lin John, Adelson P. David, Kochanek Patrick, Fink Ericka, Wisniewski Stephan, Bayir Hulya, Clark Robert S.B., Brown Danni, Bell Michael
118	GLUCOSE METABOLISM AFTER BRAIN INJURY STUDIED BY 13C-GLUCOSE AND MICRODIALYSIS	Metabolism/Physiological Assessments	TBI	Fredrik	Clausen	Uppsala University	Clausen Fredrik, Hillered Lars, Gustafsson Jan
119	CORTICAL SPREADING DEPRESSIONS ARE ASSOCIATED WITH LOW ARTERIAL AND CEREBRAL PERFUSION PRESSURES AFTER TRAUMATIC BRAIN INJURY IN HUMANS	Metabolism/Physiological Assessments, Electrophysiology	TBI	Jed	Hartings	University of Cincinnati	Hartings Jed, Manning Andrew, Fabricius Martin, Bullock Ross, Strong Anthony

120	AGE-RELATED DIFFERENCES IN THE FUNCTIONAL OUTCOMES OF PATIENTS WITH TRAUMATIC SCI: ANALYSIS OF THE THIRD NATIONAL ACUTE SPINAL CORD INJURY STUDY (NASCIS-3) DATABASE	Model Characterization/ Behavioral Measures	SCI	Julio	Furlan	Toronto Western Research Institute	Furlan Julio, Bracken Michael, Fehlings Michael
121	CHARACTERIZATION OF THE PATHOLOGICAL AND MOLECULAR CHANGES IN SPINAL CORD WHITE MATTER AND GREY MATTER IN A MODEL OF ISCHEMIA-REPERFUSION INJURY	Model Characterization/ Behavioral Measures	SCI	Yongchao	Wu	University of Toronto	Wu Yongchao, Liu Yang, Wang Jian, Yu Wenru, Fehlings Michael
122	PREVALENCE OF COMORBIDITY AND ITS ASSOCIATION WITH DISCHARGE OUTCOMES IN TRAUMATICALLY BRAIN-INJURED OLDER ADULTS	Model Characterization/ Behavioral Measures	TBI	Hilaire	Thompson	The University of Washington	Thompson Hilaire, Temkin Nancy, Dikmen Sureyya, Kagan Sarah
123	COMPARISON OF THE FIXED AND ACCELERATED SPEED VERSIONS OF ROTAROD TESTING FOR THE EVALUATION OF MOTOR DEFICITS IN AN EXPERIMENTAL RAT MODEL OF PBBI.	Model Characterization/ Behavioral Measures	TBI	Deborah	Shear	Walter Reed Army Institute of Research	Shear Deborah, Chen Zhiyong, Washington Patricia, Evangelista Clifford, Lu May, Tortella Frank
126	CONTEXTAL FEAR ENHANCEMENT FOLLOWING CONCUSSIVE INJURY IN RATS	Model Characterization/ Behavioral Measures	TBI	Maxine	Reger	UCLA	Reger Maxine, Poulos Andrew, Hovda David, Giza Christopher, Fanselow Michael
127	GENERATION AND ANALYSIS OF BLAST WAVES FROM A COMPRESSED AIR DRIVEN SHOCK TUBE	Model Characterization/ Behavioral Measures	TBI	Joseph	Atkinson	Florida Institute of Technology	Atkinson Joseph, Kirk Daniel, Gutierrez Hector, Svetlov Stanislav, Wang Kevin, Hayes Ronald
128	LONG-TERM LOCOMOTOR OUTCOME PREDICTED USING HYPER-ACUTE IN VIVO DIFFUSION TENSOR IMAGING	Model Characterization/ Behavioral Measures, Anatomy/Histology/Imaging	SCI	Joong	Kim	Washington University in St. Louis	Kim Joong H, Song Bortsong Richard, Song Sheng-Kwei
129	AN FMRI STUDY OF SELECTIVE ATTENTION DEFICITS IN SEMI-ACUTE MILD TRAUMATIC BRAIN INJURY	Model Characterization/ Behavioral Measures, Anatomy/Histology/Imaging	TBI	Andrew	Mayer	MIND Research Network	Mayer Andrew, Ling Josef, Mannel Maggie, Elgie Robert, Phillips John, Yeo Ronald, Doezeema David, Gasparovic Chuck
130	A NEW LARGE-ANIMAL MODEL OF PEDIATRIC TRAUMATIC BRAIN INJURY (TBI) WITH SECONDARY HYPOXIC INSULT	Model Characterization/ Behavioral Measures, Anatomy/Histology/Imaging	TBI	Juan	Solano	University of Miami	Solano Juan P., Luqman Ali, Ramirez Miguel, He Dansha, Padgett Kyle, Dietrich W. Dalton, Kuluz John W.
131	HIPPOCAMPAL THETA RHYTHM IS DECREASED IN RATS FOLLOWING LATERAL FLUID PERCUSSION INJURY	Model Characterization/ Behavioral Measures, Electrophysiology	TBI	Mark	Fedor	UC Davis School of Medicine	Fedor, M.D. Mark, Berman, Ph.D. Robert, Muizelaar, M.D., Ph.D. J. Paul, Lyeth, Ph.D. Bruce
132	TEMPORAL CHANGES IN BRAIN TISSUE OXYGEN AND METABOLISM FOLLOWING DIFFUSE TRAUMATIC BRAIN INJURY IN PIGLETS	Model Characterization/ Behavioral Measures, Metabolism/Physiological Assessments	TBI	Stuart	Friess	The Children's Hospital of Philadelphia	Friess Stuart, Ralston Jill, Eucker Stephanie, Helfaer Mark, Margulies Susan
133	TAT-MEDIATED INTRACELLULAR DELIVERY IS DEPENDENT UPON CELL-TYPE AND PHENOTYPE: IMPLICATIONS FOR DELIVERY TO ACTIVATED ASTROCYTES FOLLOWING INJURY	Therapeutic Intervention	In Vitro Injury	Melissa	Simon	Columbia University	Simon Melissa J, Gao Shan, Banta Scott, Morrison III Barclay
134	SURGERY FOR CENTRAL CORD SYNDROME, A RANDOMIZED STUDY	Therapeutic Intervention	SCI	Bizhan	Aarabi	University of Maryland School of Medicine	Aarabi Bizhan, Iguchi Mark, Koltz Michael, Ibrahim David, Aresco Carla, Blacklock Tiffany
135	DELAYED ADMINISTRATION OF ADV-ZFP-VEGF MAY PROMOTE ANGIOGENESIS AND REDUCE CELL DEATH FOLLOWING ACUTE SPINAL CORD INJURY IN RATS	Therapeutic Intervention	SCI	Sarah	Figley	University of Toronto	Figley Sarah, Spratt Kaye, Lee Gary, Ando Dale, Suroskey Richard, Liu Yang, Fehlings Michael
136	POST-TRAUMA TREATMENT WITH MIGLUSTAT IS NEUROPROTECTIVE IN A RAT MODEL OF CONTUSION SPINAL CORD INJURY.	Therapeutic Intervention	SCI	Ravinder	Pannu	Medical University of South Carolina	Pannu Ravinder, Singh Avtar, Singh Inderjit
137	EFFICACY OF PLATELET CONCENTRATES IN PATIENTS WITH BRAIN TRAUMA AND BLEEDING DIATHESIS.	Therapeutic Intervention	TBI	Michael	Nekludov	Karolinska Institute	Nekludov Michael, Bellander Bo-Michael
139	CASE CONTROL STUDY ON DECOMPRESSIVE CRANIECTOMY IN CHILDREN WITH TRAUMATIC BRAIN INJURY	Therapeutic Intervention	TBI	Jorn	Fierstra	Toronto: The Hospital for Sick Children	Fierstra Jorn, Grewal Sanjeev, Di Battista Ashley, Shibata Audrey, Merat Michele, Guerguerian Anne- Marie
140	SELECTIVE BRAIN COOLING ATTENUATES ELEVATED INTRACRANIAL PRESSURE INDUCED BY PENETRATING BALLISTIC-LIKE BRAIN INJURY IN RATS	Therapeutic Intervention	TBI	Guo	Wei		Wei Guo, Yang Xiaofang, Tortella Frank C, Lu Xi-Chun M

141	RESUSCITATION OF COMBINED TRAUMATIC BRAIN INJURY AND HEMORRHAGIC SHOCK WITH POLYNITROXYL ALBUMIN: EFFECT ON FLUID REQUIREMENTS, BLOOD PRESSURE, SURVIVAL AND NEUROPATHOLOGY	Therapeutic Intervention	TBI	Jennifer	Exo	University of Pittsburgh School of Medicine	Exo Jennifer, Shellington David, Vagni Vincent, Feldman Keri, Ma Li, Hsia Carlton, Clark Robert, Bayir Hulya, Jenkins Larry, Dixon C. Edward, Kochanek Patrick
142	EFFECTS OF LIPID PEROXIDATION INHIBITION ON CALPAIN-MEDIATED CYTOSKELETAL DEGRADATION AFTER TBI.	Therapeutic Intervention	TBI	Ayman	Mustafa	University of Kentucky	Mustafa Ayman, Carrico Kimberly, Thompson Stephanie, Hall Edward
143	PYRIDOXINE ADMINISTRATION IMPROVES BEHAVIORAL AND ANATOMICAL OUTCOME FOLLOWING UNILATERAL CCI IN RATS	Therapeutic Intervention	TBI	Nicholas	Kuypers	Southern Illinois University	Kuypers Nicholas, Hoane Michael
144	POTENTIAL NEUROPROTECTIVE MECHANISMS OF FACTOR VII THERAPY FOLLOWING TRAUMATIC BRAIN INJURY IN THE PIG	Therapeutic Intervention	TBI	Xiao-Han	Chen	University of Pennsylvania	Chen Xiao-Han, Tong Huaiyu, Browne Kevin, Eng Andrew, Smith Douglas
145	SPINAL CORD INJURY: A COMPARISON OF NEURAL STEM CELL AND EMBRYONIC STEM CELL TREATMENTS.	Therapeutic Intervention, Anatomy/Histology/Imaging	SCI	Daniele	Bottai	University of Milan	Bottai Daniele, Madaschi Laura, Cigognini Daniela, Adami Raffaella, Nicora Emanuela, Di Giulio Anna Maria, Gorio Alfredo
146	HYPEROXIC RESUSCITATION AFTER TRAUMATIC BRAIN INJURY IN IMMATURE RATS	Therapeutic Intervention, Anatomy/Histology/Imaging	TBI	Fasil	Mohomed	University of Maryland	Mohomed Fasil, Saraswati Manda, Fiskum Gary, Robertson Courtney
148	GLUTAMATE RECEPTOR ANTAGONISTS EXACERBATE NEURODEGENERATION, TRAUMATIC AXONAL INJURY, AND NECROTIC AND APOPTOTIC CELL DEATH IN THE IMMATURE RAT FOLLOWING CLOSED HEAD INJURY	Therapeutic Intervention, Cellular-Molecular Signaling Pathways	TBI	Jimmy	Huh	Children's Hospital Of Philadelphia	Huh Jimmy, Widing Ashley, Gin Monica, Raghupathi Ramesh
149	THE CORRELATION BETWEEN APOE POLYMORPHISM AND ELECTROENCEPHALOGRAPH OF PATIENTS WITH MILD/MODERATE TRAUMATIC BRAIN INJURY IN ACUTE PHASE	Therapeutic Intervention, Electrophysiology	TBI	Xiaochuan	Sun	First Affiliated Hospital of Chongqing Medical University	Sun Xiaochuan, He Xuezhi, Jiang Yong, Dan Wei, Wu Haitao, Guo Zongduo
150	AN IN VITRO NEUROPROTECTION ASSAY BASED ON HIGH-EFFICIENCY TRANSDUCTION OF PRIMARY CULTURED NEURONS	Therapeutic Intervention, Growth Factors/Regeneration/Plasticity	In Vitro Injury	Alisse	Hauspurg	University of Pennsylvania	Hauspurg Alisse, Malik Saafan, Watson Deborah
151	TRANSPLANTATION OF CNTF-EXPRESSING ADULT OLIGODENDROCYTE PRECURSOR CELLS PROMOTES REMYELINATION AND FUNCTIONAL RECOVERY AFTER SPINAL CORD INJURY.	Therapeutic Intervention, Growth Factors/Regeneration/Plasticity	SCI	Qilin	Cao	Kentucky Spinal Cord Injury Research Center	Cao Qilin, He Qian, Cheng Xiaoxin, Wang Yaping, Howard Russell M, Zhang Yi-Ping, Shields Christopher B, Magnuson David SK, Whittemore Scott R
152	ADULT HUMAN NEURAL STEM/PROGENITOR CELLS ISOLATED FROM NEUROSURGICAL RESECTION TISSUES AS A CELL SOURCE FOR TRANSPLANTATION FOLLOWING TBI	Therapeutic Intervention, Growth Factors/Regeneration/Plasticity	TBI	Dong	Sun	Virginia Commonwealth University	Sun Dong, Gugliotta Marinella, Fillmore Helen, Hu Wenhui, Holloway Katherine, Bullock Ross, Young Harold
154	THE NALP1 INFLAMMASOME IN CORTICAL NEURONS REGULATES INFLAMMATION FOLLOWING TRAUMATIC BRAIN INJURY	Therapeutic Intervention, Inflammation/Neurotoxicity	TBI	Juan Pablo	de Rivero Vaccari	University of Miami Miller School of Medicine	de Rivero Vaccari Juan Pablo, Lotocki George, Alonso Ofelia F., Bramlett Helen M., Dietrich W. Dalton, Keane Robert W.
155	PARP INHIBITION IMPROVES RECOVERY AND REDUCES MICROGLIAL ACTIVATION AFTER TRAUMATIC BRAIN INJURY	Therapeutic Intervention, Inflammation/Neurotoxicity	TBI	Kimberly	Byrnes	Georgetown University	Byrnes Kimberly, Stoica Bogdan, Faden Alan
156	CEREBROSPINAL FLUID DRAINAGE AND PRESSURE MONITORING AFTER ACUTE HUMAN SPINAL CORD INJURY	Therapeutic Intervention, Metabolism/Physiological Assessments	SCI	Brian	Kwon	University of British Columbia	Kwon Brian, Belanger Lise, Chan Donna, Bernardo Arlene, Slobogean Gerard, Boyd Michael, Paquette Scott, Umedaly Hamed, Giffin Mitch, Street John, Fisher Charles, Curt Armin, Dvorak Marcel
157	EFFECT OF SHORT PERIODS OF NORMOBARIC HYPEROXIA ON LOCAL BRAIN TISSUE OXYGENATION AND CEREBROSPINAL FLUID OXIDATIVE STRESS MARKERS IN SEVERE TRAUMATIC BRAIN INJURY	Therapeutic Intervention, Metabolism/Physiological Assessments	TBI	Ava	Puccio	University of Pittsburgh	Puccio Ava, Hoffman Leslie, Bayir Hulya, Zullo Thomas, Fischer Michael, Darby Joseph, Alexander Sheila, Dixon C. Edward, Okonkwo David, Kochanek Patrick

158	THERAPEUTIC HYPOTHERMIA EXACERBATES INCREASES IN CEREBROSPINAL FLUID ENDOTHELIN-1 LEVELS IN INFANTS AND CHILDREN AFTER SEVERE TRAUMATIC BRAIN INJURY	Therapeutic Intervention, Metabolism/Physiological Assessments	TBI	Rosanne	Salonia	University of Pittsburgh School of Medicine	Salonia Rosanne, Polyac Samuel, Ruppel Randall, Klamerus Megan, Feldman Keri, Adelson P. David, Brown Danielle, Berger Rachel, Bell Michael, Fink Ericka, Clark Robert, Bayir Hulya, Kochanek Patrick
159	THE NEUROPROTECTIVE EFFECT OF POST INJURY ADMINISTRATION OF RECOMBINANT HUMAN ERYTHROPOIETIN (RHUEPO) AFTER CORTICAL IMPACT INJURY IN RATS	Therapeutic Intervention, Metabolism/Physiological Assessments	TBI	Leela	Cherian	Baylor College of Medicine	Cherian Leela, Robertson Claudia
160	APPLICATION OF MULTIVARIATE METHODS FOR ANALYSIS OF COMPLEX PHYSIOLOGICAL DATA IN THE NEUROINTENSIVE CARE UNIT AFTER HEMORRHAGIC SHOCK AND TRAUMATIC BRAIN INJURY	Therapeutic Intervention, Metabolism/Physiological Assessments	TBI	Mitchell	Cohen	University of California, San Francisco	Ferguson Adam, Cohen Mitchell, Morabito Diane, Manley Geoff
161	EFFECTS OF ACETYL-L-CARNITINE ON MITOCHONDRIAL DYSFUNCTION FOLLOWING ACUTE CONTUSION SPINAL CORD INJURY	Therapeutic Intervention, Model Characterization/ Behavioral Measures	SCI	Samirkumar	Patel	University of Kentucky	Patel Samir, Lyttle Travis, Sullivan Patrick, Rabchevsky Alexander
162	ACUTE, SUBCHRONIC, OR CHRONIC TREATMENTS DIFFERENTIALLY INFLUENCE DEVELOPMENT OF SCI SPASTICITY.	Therapeutic Intervention, Model Characterization/ Behavioral Measures	SCI	Floyd	Thompson	University of Florida	Thompson Floyd, Hou Jiamei, Parmer Ron, Cheng Yanping, Jain Rita, Bose Prodip
163	EVALUATING THE RELATION OF CORTISOL LEVELS, GENDER, AND DEPRESSION IN TRAUMATIC BRAIN INJURY PATIENTS	Therapeutic Intervention, Model Characterization/ Behavioral Measures	TBI	Emily	Rogers	University of Pittsburgh, Emory University	Rogers Emily H., Loucks Tammy L., Fabio Anthony, Hines Alicia, Yuan Zhifang, Dixon C. Edward, Berga Sarah L., Wagner Amy K.
164	THE EFFECTS OF ACUTE VOLUNTARY MOTOR ENRICHMENT ON RECOVERY OF FUNCTION FOLLOWING MEDIAL FRONTAL CORTEX CONTUSIONS IN RATS	Therapeutic Intervention, Model Characterization/ Behavioral Measures	TBI	Kyle	Fink	University of Portland	Fink Kyle, Crane Andrew, Smith Jeffrey

**JULY 27 - 29, 2008
HILTON IN THE WALT DISNEY WORLD RESORT
LAKE BUENA VISTA, FLORIDA**

POSTER SESSION C – TUESDAY 9:45 – 11:00



P#	TITLE	ASPECTS	AREA	FIRST	LAST	INSTITUTION	ALL AUTHORS
166	MAGNETIC RESONANCE TRACKING OF NOVEL BIODEGRADABLE, BIOCOMPATIBLE PLGA CHANNELS FOR SPINAL CORD REPAIR	Anatomy/Histology/Imaging	SCI	Xuefen	Yang	University of Ottawa, Ottawa Health Research Institute	Yang Xuefen, Westwick Harrison, Cao Xudong, Tsai Eve
167	MRI BIOMARKERS OF TRAUMATIC BRAIN INJURY: IDENTIFICATION OF DISTINCT INJURY SUBTYPES AND CORRELATION WITH FUNCTIONAL OUTCOME.	Anatomy/Histology/Imaging	TBI	Ramon	Diaz-Arrastia	University of Texas Southwestern Medical Center	Diaz-Arrastia Ramon, Peng Lifang, Ding Kan, Redfern Shelley, Ayala Rebecca, Moore Carol, Harper Caryn, Madden Christopher, Devous Michael
168	IMPROVING THE DETECTION OF DIFFUSE AXONAL INJURY BY COMPLEMENTARY USE OF ADVANCED MRI	Anatomy/Histology/Imaging	TBI	Zhifeng	Kou	Wayne State University	Kou Zhifeng, Benson Randall, Gattu Ramtilak, Haacke E Mark
169	HIPPOCAMPAL CYTOARCHITECTURAL RECOVERY FOLLOWING EXPERIMENTAL TBI IN THE IMMATURE POSTNATAL DAY (PND) 7 RAT	Anatomy/Histology/Imaging	TBI	Glenn	Gobbel	University of Pittsburgh	Gobbel Glenn T., Srinivas Ravi, Fellows-Mayle Wendy, Adelson P. David
170	SYNAPTIC PLASTICITY IN A RODENT MODEL OF DIFFUSE TRAUMATIC BRAIN INJURY	Anatomy/Histology/Imaging	TBI	Patrick	Schafer	Wayne State University	Schafer Patrick, Kreipke Christian, Rafols Jose
171	A COMPUTATIONAL INVESTIGATION INTO THE PATTERNS OF GLUTAMATE RECEPTOR ACTIVATION FOLLOWING PHYSIOLOGIC AND INJURY INDUCED GLUTAMATE RELEASE	Cellular-Molecular Signaling Pathways	In Vitro Injury	Pallab	Singh	University of Pennsylvania	Singh Pallab, Meaney David
172	IN VITRO ASTROCYTIC RESPONSES ON DIFFERENT MAGNITUDES OF SHORT DURATION OVERPRESSURE	Cellular-Molecular Signaling Pathways	In Vitro Injury	Lai Yee	Leung	Wayne State University	Leung Lai Yee, Mao Li, VandeVord Pamela
173	SECONDARY PEAKS IN SERUM S100B IN HUMANS FOLLOWING TRAUMATIC BRAIN INJURY IS NOT RELATED TO MONITORED SECONDARY INSULTS	Cellular-Molecular Signaling Pathways	TBI	Eric	Thelin	Uppsala University	Thelin Eric, Bellander Bo-Michael
174	CHANGES IN THE EXPRESSION LEVEL OF TWO ISOFORMS OF STRIATAL ENRICHED PHOSPHATASE (STEP) FOLLOWING CONTROLLED CORTICAL IMPACT (CCI)	Cellular-Molecular Signaling Pathways	TBI	Mahlet	Mesfin	University of Pennsylvania	Mesfin Mahlet N., Creed Jennifer, Raghupathi Ramesh, Meaney David F.
175	THE EFFECTS OF TRAUMATIC BRAIN INJURY ON CYCLIN-DEPENDENT KINASE 5 (CDK5) AND P35/P25 EXPRESSION IN RAT HIPPOCAMPUS	Cellular-Molecular Signaling Pathways	TBI	Hong	Yan	University of Pittsburgh	Yan Hong, Ma Xiecheng, Li Youming, Dixon Edward
177	TRANSCRIPTOMIC AND PROTEOMIC ANALYSIS OF SPINAL MICROVASCULAR ENDOTHELIAL PLASTICITY FOLLOWING FOCAL ISCHEMIC SPINAL CORD INJURY (SCI)	Cellular-Molecular Signaling Pathways, Anatomy/Histology/Imaging	SCI	Toros	Dincman	Univ of Louisville	Benton Richard, Maddie Melissa, Whitemore Scott, Dincman Toros
178	IMPAIRED AXONAL TRANSPORT AND INTRA-AXONAL JNK ACTIVATION FOLLOWING CONCUSSIVE BRAIN INJURY IN THE ADULT MOUSE.	Cellular-Molecular Signaling Pathways, Anatomy/Histology/Imaging	TBI	Jennifer	Creed	Drexel University College of Medicine	Creed Jennifer, Sutter Marijke, Raghupathi Ramesh
179	TRANSIENT INDUCTION OF IGF-1/IGF-1R SIGNALING IN THE MOUSE BRAIN FOLLOWING TRAUMATIC BRAIN INJURY	Cellular-Molecular Signaling Pathways, Growth Factors/Regeneration/Plasticity	TBI	Shaun	Carlson		Madathil Sindhu Kizhakke, Carlson Shaun W., Foozer Heather, Saatman Kathryn
180	ROLE OF NRF2 IN PROTECTION AGAINST TRAUMATIC BRAIN INJURY IN MICE	Cellular-Molecular Signaling Pathways, Inflammation/Neurotoxicity	TBI	Wei	Jin	China	Jin Wei, Wang Handong, Yan Wei, Hu Zhigang, Chen Gang, Yin Hongxia
181	CHANGES IN CYTOSOLIC GAPDH ACTIVITY FOLLOWING CONTROLLED CORTICAL IMPACT IN JUVENILE AND ADULT RAT BRAINS.	Cellular-Molecular Signaling Pathways, Metabolism/Physiological Assessments	TBI	Ying	Deng-Bryant	ULCA	Deng-Bryant Ying, Appelberg Sofia, Hovda David, Prins Mayumi
182	NON-CONVULSIVE SEIZURE PROFILES IN RATS IDENTIFIED USING CONTINUOUS EEG MONITORING FOLLOWING A PENETRATING BALLISTIC-LIKE BRAIN INJURY (PBBi)	Electrophysiology	TBI	May	Lu	Walter Reed Army Institute of Research	Lu Xi-Chun May, Si Yuanzheng, Cao Ying, Hartings Jed, Wei Guo, Yang Xiaofang, Tortella Frank
183	EXTRAMEDULLARY CHITOSAN CHANNELS PROMOTE SURVIVAL OF TRANSPLANTED NEURAL STEM/PROGENITOR CELLS AND CREATE A TISSUE BRIDGE AFTER COMPLETE SPINAL CORD TRANSECTION	Growth Factors/Regeneration/Plasticity	SCI	Hiroshi	Nomura	Hiroshima Red Cross Hospital	Nomura Hiroshi, Zahir Tasneem, Kim Howard, Kulbatski Iris, Morshead Cindi, Iwamoto Yukihide, Shoichet Molly, Tator Charles
184	THE INHIBITION OF EGFR BY PEPTIDE DRUG SYNTHESIZED IN SILICO.	Growth Factors/Regeneration/Plasticity	SCI	Tomonori	Endo	Chiba University	Endo Tomonori, Tamura Yutaka, Koda Masao, Yamazaki Masashi, Okawa Akihiko, Hashimoto Masayuki, Fujiyoshi Takayuki, Kawabe Junko, Hayashi Kouichi, Furuya Takeo, Takahashi Kazuhisa
185	DIRECT ISOLATION OF NEURAL STEM CELLS IN THE ADULT HIPPOCAMPUS AFTER TRAUMATIC BRAIN INJURY	Growth Factors/Regeneration/Plasticity	TBI	Jinhui	Chen		Gao Xiang, Chen Jinhui

186	PHOSPHACAN AND GELATINASE: POTENTIAL MEDIATORS OF WHITE MATTER INJURY AND RECOVERY FOLLOWING TRAUMATIC BRAIN INJURY	Growth Factors/ Regeneration/Plasticity	TBI	Linda	Phillips		Phillips Linda, Harris Lesley, Black Raiford, Lee Nancy, Reeves Thomas
187	MINIMALLY INVASIVE INTRATHECAL DELIVERY OF NEURAL STEM/PROGENITOR CELLS FOR SPINAL CORD REPAIR	Growth Factors/ Regeneration/Plasticity, Anatomy/Histology/Imaging	SCI	Andrea	Mothe	Toronto Western Research Institute	Mothe Andrea, Bozkhurt Gokhan, Wang Xing-Hua, Keating Armand, Tator Charles
188	RECOVERY DEPENDENT DIFFERENCES IN MEMBRANE-TYPE 5 MATRIX METALLOPROTEINASE AND N-CADHERIN EXPRESSION DURING INJURY-INDUCED SYNAPTOGENESIS	Growth Factors/ Regeneration/Plasticity, Electrophysiology	TBI	Kelly	Warren	Virginia Commonwealth University	Warren Kelly, Lee Nancy, Black Raiford, Reeves Thomas, Phillips Linda
189	CALPAIN AND CASPASE MEDIATED PROTEOLYSIS OF AII-SPECTRIN AND BII-SPECTRIN IN RAT CEREBROCORTEX CULTURE UNDER ONCOTIC, APOPTOTIC AND EXCITOTOXIC CHALLENGES	Growth Factors/ Regeneration/Plasticity, Inflammation/Neurotoxicity	In Vitro Injury	Wenrong	Zheng	Banyan Biomarkers Inc.	Zheng Wenrong, Liu Ming Cheng, Hayes Ronald L., Wang Kevin K.W.
192	ADENOSINE A1 RECEPTOR ACTIVATION AS A BRAKE ON NEUROINFLAMMATION AFTER EXPERIMENTAL TRAUMATIC BRAIN INJURY IN MICE	Inflammation/Neurotoxicity	TBI	Patrick	Kochanek	University of Pittsburgh School of Medicine	Haselkorn M. Lee, Shellington David, Jackson Edwin, Vagni Vincent, Feldman Keri, Dubey Raghvendra, Gillespie Delbert, Bell Michael, Clark Robert, Jenkins Larry, Schnermann Jurgen, Homanics Gregg, Kochanek Patrick
193	WHOLE BODY BLAST INJURY: CENTRAL NERVOUS SYSTEM RESPONSE	Inflammation/Neurotoxicity, Anatomy/Histology/Imaging	TBI	Paul	Tompkins	University of Oklahoma, HSC	Tompkins Paul, Tesiram Yasvir, Lerner Megan, Brackett Daniel, Lightfoot Stan
194	INFLAMMATORY CYTOKINE EXPRESSION AND THE DEVELOPMENT OF INJURY-SEVERITY BIOMARKERS AFTER HUMAN SPINAL CORD INJURY	Inflammation/Neurotoxicity, Metabolism/Physiological Assessments	SCI	Brian	Kwon	University of British Columbia	Kwon Brian, Stammers Anthea, Belanger Lise, Chan Donna, Bernardo Arlene, Umedaly Hamed, Giffin Mitch, Paquette Scott, Boyd Michael, Street John, Fisher Charles, Dvorak Marcel
195	ALPHA-II SPECTRIN BREAKDOWN PRODUCT KINETICS IN ACUTE BRAIN INJURY	Metabolism/Physiological Assessments	Ischemic-Hypoxic Injury	Gretchen	Brophy	Virginia Commonwealth University	Brophy Gretchen, Papa Linda, Liu Ming Cheng, Wang Kevin, Hayes Ronald, Lewis Stephen
196	CEREBRAL HEMODYNAMIC PREDICTORS OF POOR LONG TERM OUTCOME IN SEVERE PEDIATRIC TRAUMATIC BRAIN INJURY	Metabolism/Physiological Assessments	TBI	Monica	Vavilala	University of Washington	Chaiwat Onuma, Sharma Deepak, Udomphorn Yuthana, Armstead William, Vavilala Monica
197	WHOLE-BRAIN IMAGING OF PROTON METABOLITE CHANGES IN MILD-TO-MODERATE TRAUMATIC BRAIN INJURY	Metabolism/Physiological Assessments	TBI	Jonathan	Jagid	University of Miami	Jagid Jonathan, Govind Varan, Gold Stuart, Saigal Gaurav, Harris Leo, Maudsley Andrew
198	PLASMA OXIDATION-REDUCTION POTENTIAL IN TRAUMATIC BRAIN INJURY	Metabolism/Physiological Assessments	TBI	David	Bar-Or	Swedish Medical Center	Bar-Or David, Rael Leonard, Bar-Or Raphael, Mains Charles, Slone Denetta, Levy A. Stewart
199	AUTOMATED HIGH THROUGHPUT AXON INJURY SYSTEM FOR THE STUDY OF BRAIN INJURY MECHANISMS	Model Characterization/ Behavioral Measures	In Vitro Injury	Bryan	Pfister	New Jersey Institute of Technology	Guo Yi, Chen Linda, Pfister Bryan
200	ATTITUDES TOWARD ELDERLY INDIVIDUALS WITH NEUROTRAUMA AMONG HEALTHCARE PROFESSIONALS AND NEUROSCIENTISTS	Model Characterization/ Behavioral Measures	SCI	Julio	Furlan	Toronto Western Research Institute	Furlan Julio, Fehlings Michael
201	INSTRUMENTED HEADGEAR TO STUDY HEAD KINEMATICS IN INFANTS AND TODDLERS	Model Characterization/ Behavioral Measures	TBI	Patricia	Quebada	Dartmouth	Quebada Patricia, Greenwald Rick, Buck Aaron, Eypper Dave, Duhaime Ann-Christine
202	RELIABILITY AND VALIDITY OF THE NEUROPHYSICAL OUTCOME SCALE FOR USE IN TRAUMATIC BRAIN INJURY	Model Characterization/ Behavioral Measures	TBI	Elisabeth	Wilde	Baylor College of Medicine	Wilde Elisabeth, McCauley Stephen, Kelly Tara, Weyand Annie, Yallampalli Ragini, Waldron Eric, Boake Corwin, Levin Harvey, Clifton Guy, Valadka Alex, Moretti Paolo
203	KINEMATICS OF THE INFANT AND TODDLER HEAD DURING LOW HEIGHT FALLS	Model Characterization/ Behavioral Measures	TBI	Nicole	Ibrahim	University of Pennsylvania	Ibrahim Nicole, Coats Brittany, Margulies Susan
204	BLAST INDUCED TRAUMATIC BRAIN INJURY: CHARACTERIZING THE INJURY PATHOLOGY	Model Characterization/ Behavioral Measures	TBI	Ryan	Readnower	University of Kentucky	Readnower Ryan, McCarron Richard, Chavko Mikulas, Sullivan Patrick
205	KINEMATIC ANALYSIS OF SENSORIMOTOR DEFICITS AND COMPENSATORY MECHANISMS FOLLOWING TRAUMATIC BRAIN INJURY IN RODENTS	Model Characterization/ Behavioral Measures	TBI	Connie	Myerson	University of Miami	Myerson Connie, Toth Eniko, Wasserman Joseph, Bramlett Helen, Dietrich W. Dalton, Green Edward

207	QUALITATIVE AND QUANTITATIVE MRI PARAMETERS ASSOCIATED WITH NEUROLOGICAL IMPROVEMENT IN PATIENTS WITH ACUTE CERVICAL TRAUMATIC SPINAL CORD INJURY	Model Characterization/ Behavioral Measures, Anatomy/Histology/Imaging	SCI	Julio	Furlan	Toronto Western Research Institute	Furlan Julio, Aarabi Bizhan, Fehlings Michael
208	IN SEARCH OF TRANSLATIONAL MEASURES OF SCI: MULTIVARIATE MINING OF EXISTING EXPERIMENTAL DATA TO IDENTIFY WHAT WE KNOW AND DON'T KNOW ABOUT THE EXISTENCE OF A COMMON SYNDROME FOLLOWING SCI	Model Characterization/ Behavioral Measures, Anatomy/Histology/Imaging	SCI	Adam	Ferguson	UCSF/OSU	Ferguson Adam, Irvine Karen- Amanda, Gensel John, Lin Amity, Ly Johnathan, Bresnahan Jacqueline, Beattie Michael
209	PREDICTION OF COGNITIVE SEQUELAE BASED ON ABNORMAL CT FINDINGS IN CHILDREN FOLLOWING MILD TBI	Model Characterization/ Behavioral Measures, Anatomy/Histology/Imaging	TBI	Harvey	Levin		Levin Harvey, Luerksen Thomas, Ewing-Cobbs Linda, Hanten Gerri, Dennis Maureen, Chapman Sandra
210	SCALED CORTICAL CONTUSION IN IMMATURE SWINE: EFFECT OF AGE AND GENDER ON LESION VOLUME	Model Characterization/ Behavioral Measures, Anatomy/Histology/Imaging	TBI	Ann-Christine	Duhaime	Dartmouth Hitchcock Medical Center	Duhaime Ann-Christine, Dodge Carter, Lee Ying-Lung, Quebada Patricia, Curtis Rachel, Hillier Simon, Simoni Michael, Adams Leslie, Costine Beth
211	EEG CHANGES FOLLOWING PENETRATING BALLISTIC-LIKE BRAIN INJURY IN RATS: A POWER SPECTRAL ANALYSIS	Model Characterization/ Behavioral Measures, Electrophysiology	TBI	Yuanzheng	Si	Walter Reed Army Institute of Research	Si Yuanzheng, Wei Guo, Chao Ying, Lu Xi-Chun May, Yang Xiaofang, Tortella Frank C., Hartings Jed A.
212	REGIONAL CEREBRAL OXYGENATION MONITORING USING MULTIPLE PROBES: TECHNICAL ASPECTS AND CLINICAL IMPLICATIONS.	Model Characterization/ Behavioral Measures, Metabolism/Physiological Assessments	TBI	Nicholas	Phan	UCSF	Phan Nicholas, Rosenthal Guy, Morabito Diane, Manley Geoffrey
213	ACCURACY OF SENSORY AXONAL REINNERVATION IS MEDIATED BY POST-TRAUMATIC NGF GENE THERAPY IN THE ADULT FEMORAL NERVE	Therapeutic Intervention	PNS Injury	Xinhua	Hu	University of Kentucky	Hu Xinhua, Cai Jie, Yang Jun, Smith George
214	PREOPERATIVE AND POSTOPERATIVE ADMINISTRATION OF PHOSPHODIESTERASE INHIBITOR RATHER EXACERBATE LOCOMOTOR RECOVERY IN A SPINAL CORD CONTUSION INJURY IN RATS.	Therapeutic Intervention	SCI	Koichi	Hayashi	Chiba university	Hayashi Koichi, Hashimoto Masayuki, Koda Masao, Yamazaki Masashi, Okawa Akihiko, Kawabe Junko, Fujiyoshi Takayuki, Endo Tomonori, Furuya Takeo, Takahashi Kazuhisa
215	SUPPRESSION OF GLIAL SCAR INHIBITION BY PROMOTING DEGRADATION OF EXTRACELLULAR MATRIX AFTER CHRONIC SPINAL CORD INJURY IN RATS	Therapeutic Intervention	SCI	Junko	Kawabe	Chiba University	Kawabe Junko, Koda Masao, Hashimoto Masayuki, Fujiyoshi Takayuki, Hayashi Koichi, Furuya Takeo, Endo Tomonori, Okawa Akihiko, Yamazaki Masashi, Takahashi Kazuhisa
216	ADMINISTRATION OF BCL-XL FUSION PROTEIN IS PROTECTIVE AFTER SPINAL CORD INJURY IN A RAT MODEL	Therapeutic Intervention	SCI	Candace	Floyd	University of Alabama at Birmingham	Dunlap Malisa J., Hall Alicia M., Floyd Candace L.
217	ACUTE ENDOCRINE RESPONSE FOLLOWING SEVERE TRAUMATIC BRAIN INJURY IN CHILDREN	Therapeutic Intervention	TBI	Ravi	Srinivas	University of Pittsburgh	Srinivas Ravi, Brown Danielle, Chang Yuefang, Adelson P. David
218	LEVETIRACETAM VERSUS PHENYTOIN AS SEIZURE PROPHYLAXIS IN SEVERE TRAUMATIC BRAIN INJURY	Therapeutic Intervention	TBI	Kristen	Jones	University of Pittsburgh	Jones Kristen E, Puccio Ava M, Hashman Kathy J, Jankowitz Brian T, Fischer Michael, Okonkwo David O
219	OUR EXPERIENCE OF SURGICAL INTERVENTION IN GERIATRIC HEAD TRAUMA	Therapeutic Intervention	TBI	Virendra	Sinha	S.M.S.Medical College	D.Sinha Virendra, Chopra Sanjiv, Gupta Vishnu, Bagaria H.R.
220	EFFECTS OF HYPOTHERMIA TREATMENT AFTER EXPERIMENTAL TRAUMATIC BRAIN INJURY IN THE IMMATURE POSTNATAL DAY (PND) 17 RAT	Therapeutic Intervention	TBI	P. David	Adelson	University of Pittsburgh	Adelson P. David, Srinivas Ravi, Fellows-Mayle Wendy, Gobbel Glenn T.
221	EFFECTS OF MAGNESIUM THERAPY AND ENRICHED ENVIRONMENT ON TRAUMATIC BRAIN INJURY	Therapeutic Intervention	TBI	Darren	Miller	University of Portland	Miller Darren, Morgart Erin, Smith Jeffrey
222	FLUMAZENIL ADMINISTRATION ATTENUATES MORRIS WATER MAZE DYSFUNCTION IN THE IMMATURE POSTNATAL DAY (PND) 7 RAT FOLLOWING CONTROLLED CORTICAL IMPACT	Therapeutic Intervention	TBI	Pawel	Ochalski	University of Pittsburgh	Ochalski Pawel G., Fellows- Mayle Wendy, Hsieh Lily B., Srinivas Ravi, Okonkwo David O., Dixon C. Edward, Adelson P. David
223	COG1410, AN APOLIPOPROTEIN E-BASED PEPTIDE, IMPROVES COGNITIVE PERFORMANCE AND REDUCES CORTICAL LOSS FOLLOWING MODERATE FLUID PERCUSSION INJURY IN THE RAT.	Therapeutic Intervention	TBI	Nicholas	Kaufman	Southern Illinois University Carbondale	Kaufman Nicholas, Beare Jason, Tan Arlene, McKenna Suzanne, Vitek Michael, Hoane Michael

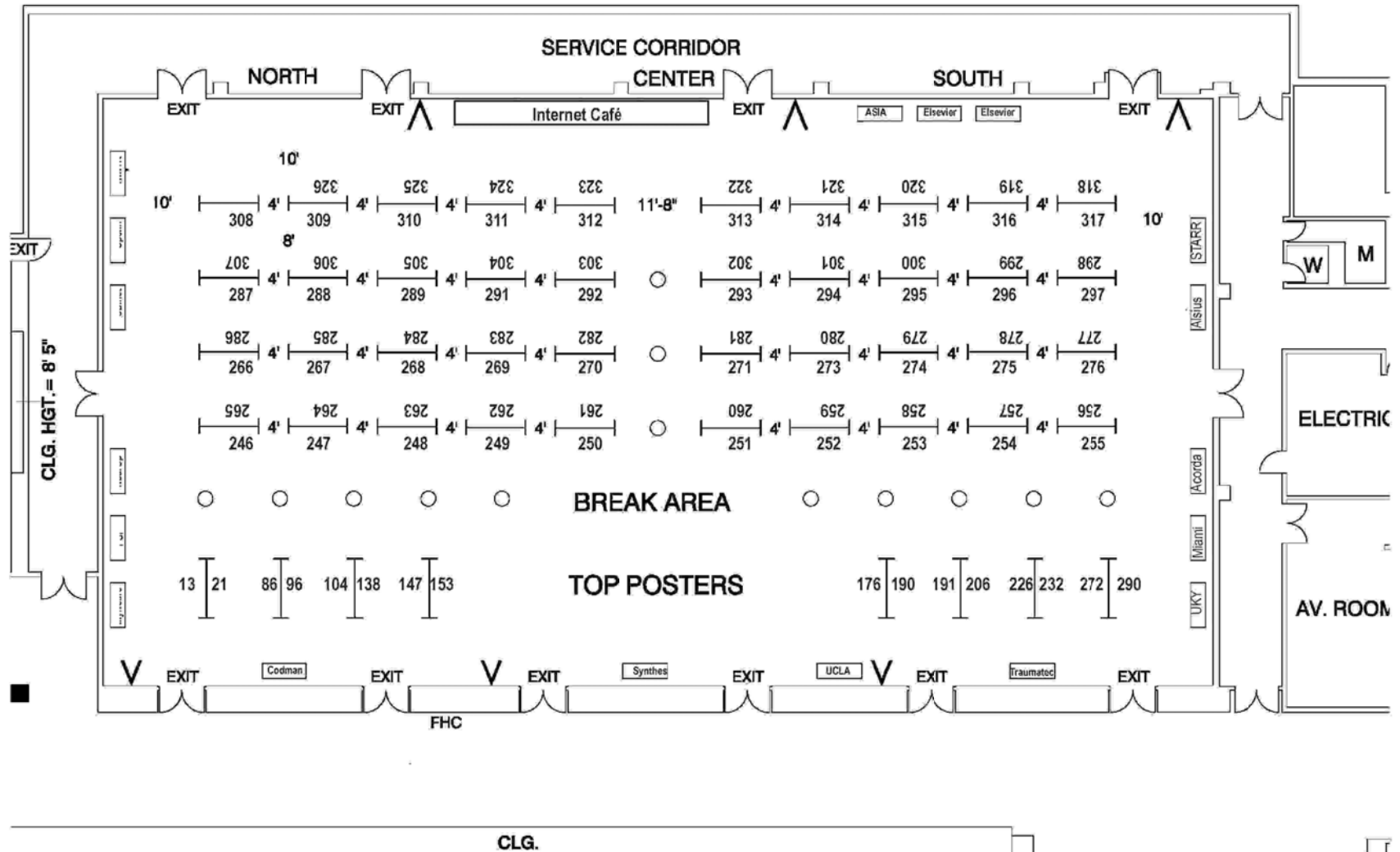
224	EXTERNAL VALIDATION OF HEAD CT RULES IN ELDERLY PATIENTS WITH MINOR HEAD INJURY	Therapeutic Intervention, Anatomy/Histology/Imaging	TBI	Sang Do	Shin	Seoul National University Hospital	Shin Sang Do, Ro Young Sun, Song Kyoung Jun, Suh Gil Joon, Kim Yu Jin, Ahn Ki Ok
225	AGE INFLUENCES THE EFFICACY OF NICOTINAMIDE, A NOVEL NEUROPROTECTANT, IN THE TREATMENT OF TRAUMATIC BRAIN INJURY FOLLOWING CONTROLLED CORTICAL IMPACT	Therapeutic Intervention, Anatomy/Histology/Imaging	TBI	Alicia	Swan	Southern Illinois	Swan Alicia, Hoane Michael
227	TARGETING ERK2 WITH LENTIVIRAL SHRNA IMPROVES TISSUE SPARING AND LOCOMOTOR FUNCTION AFTER SPINAL CORD INJURY	Therapeutic Intervention, Cellular-Molecular Signaling Pathways	SCI	Chen-Guang	Yu	University of Kentucky	Yu Chen-Guang, Yeziarski Robert P, Joshi Aashish, Raza Kashif, Li Yanzhang, Geddes James W
228	NEUROPROTECTIVE EFFECT OF HYPERBARIC OXYGEN THERAPY IN BRAIN INJURY IS MEDIATED BY PRESERVATION OF MITOCHONDRIAL MEMBRANE PROPERTIES	Therapeutic Intervention, Cellular-Molecular Signaling Pathways	TBI	Jean	Soustiel	Faculty of Medicine, Technion Israel Institute of Technology	Soustiel Jean Francois, Palzur Eilam
229	TARGETED DELETION OF THE NEURAL SPECIFIC JNK ISOFORM (JNK3) AMELIORATES POST-TARUMATIC ACTIVATION OF CASPASE-3 AND -9 AND REDUCES IMPAIRED AXONAL TRANSPORT IN THE THALAMUS	Therapeutic Intervention, Cellular-Molecular Signaling Pathways	TBI	Pushpinder Kaur	Multani	Drexel University College of Medicine	Multani Pushpinder Kaur, Creed Jennifer, Vingless Veronica, Raghupathi Ramesh
230	THE RELATIONSHIP BETWEEN SLEEP ENVIRONMENT, SLEEP DEPRIVATION, AND COGNITIVE RECOVERY AFTER TRAUMATIC BRAIN INJURY -- A REVIEW OF THE LITERATURE.	Therapeutic Intervention, Electrophysiology	TBI	Rodney	Samuelson	Virginia Commonwealth University	Samuelson Rodney, Morrison Alex, Atwal Gursant, Graham Scott, Young Harold
231	CONSTRAINT-INDUCED MOVEMENT THERAPY FOR SPINAL CORD HEMISECTION INJURY IN ADULT RATS	Therapeutic Intervention, Growth Factors/Regeneration/Plasticity	SCI	Masao	Koda	Chiba Aoba Municipal Hospital	Koda Masao, Hashimoto Masayuki, Kawabe Junko, Fujiyoshi Takayuki, Hayashi Koichi, Furuya Takeo, Endo Tomonori, Okawa Akihiko, Yamazaki Masashi
233	INFLAMMATORY FACTORS PRODUCED BY ATP-ACTIVATED MICROGLIA ARE REDUCED BY HYPOTHERMIA	Therapeutic Intervention, Inflammation/Neurotoxicity	In Vitro Injury	Tomohiro	Matsui	Yamaguchi University Graduate School of Medicine	MATSUI Tomohiro, MIURA Daisuke, KATO Yukiko, INOMOTO Takafumi, MOTOKI Yukari
234	COMPREHENSIVE EXAMINATION OF SECONDARY INJURY AND POTENTIAL SINGLE AND COMBINATORIAL NEUROPROTECTIVE THERAPEUTIC STRATEGIES	Therapeutic Intervention, Inflammation/Neurotoxicity	SCI	Cassie	Mitchell	Georgia Inst. Technology/Emory University	Mitchell Cassie, Lee Robert
235	PPAR-GAMMA ACTIVATION IS NEUROPROTECTIVE AFTER TBI BY PREVENTING INFLAMMATION AND OXIDATIVE STRESS	Therapeutic Intervention, Inflammation/Neurotoxicity	TBI	Raghu	Vemuganti	University of Wisconsin	Yi John, Brooks Nathaniel, Park Seung-Won, Dharap Ashutosh, Vemuganti Raghu
236	SIMVASTATIN TREATMENT REDUCES MICROGLIAL ACTIVATION, SYNAPTIC LOSS, AND BETA-AMYLOID LEVELS AFTER TRAUMATIC BRAIN INJURY	Therapeutic Intervention, Inflammation/Neurotoxicity	TBI	Eric	Abrahamson	University of Pittsburgh	Abrahamson Eric, Ikonovic Milos, Dixon C. Edward, DeKosky Steven
237	A TISSUE ENGINEERED SCAFFOLD SEEDED WITH HUMAN NEUROEPITHELIAL CELLS FOR TREATMENT OF SPINAL CORD INJURY	Therapeutic Intervention, Metabolism/Physiological Assessments	SCI	Michelle	LaPlaca	Georgia Institute of Technology / Emory University	Rahmani Yasmin, Simon Crystal, Schumm Michael, Sturkie Carla, West Franklin, Stice Steve, LaPlaca Michelle
238	OUTCOME PREDICTION IN SEVERE TRAUMATIC BRAIN INJURY: DOES CONVENTIONAL CT MATTER?	Therapeutic Intervention, Metabolism/Physiological Assessments	TBI	Andras	Buki	Pécs University, Pécs, Hungary	Ursprung Zsuzsanna, Czeiter Endre, Sandor Janos, Kovacs Noemi, Ezer Erzsébet, Doczi Tamas, Buki Andras
239	DECOMPRESSIVE CRANIECTOMY FOR ICP RELIEF IN SEVERE TRAUMATIC BRAIN INJURY: COMPARISON OF CEREBRAL INDICES OF BLOOD FLOW AND METABOLISM IN OPERATED AND NON-OPERATED PATIENTS.	Therapeutic Intervention, Metabolism/Physiological Assessments	TBI	Jean	Soustiel	Faculty of Medicine, Technion Israel Institute of Technology	Soustiel Jean Francois, Shik Venyamin, Mahamid Eugenia, Sviri Gil, Zaaroor Menashe
240	MILD IMPROVEMENT IN CEREBRAL GLUCOSE METABOLISM WITH VAGUS NERVE STIMULATION AFTER FLUID PERCUSSION BRAIN INJURY IN RATS.	Therapeutic Intervention, Metabolism/Physiological Assessments	TBI	Richard	Sutton	UCLA	Sutton Richard L, Ghavim Sima, Moro Nobuhiro, Tan Arlene, Smith Douglas C, Hovda David A
241	SERUM MARKERS IN A PIGLET SCALED CORTICAL IMPACT MODEL: EFFECT OF AGE AND GENDER	Therapeutic Intervention, Metabolism/Physiological Assessments	TBI	Patricia	Quebada	Dartmouth	Quebada Patricia, Lee Ying-Lung, Dodge Carter, Hillier Simon, Simoni Michael, Costine Beth, Duhaime Ann-Christine
242	DECREASED GLIAL ACTIVATION AND IL-1 B PRODUCTION WITH IL-1 RECEPTOR ANTAGONIST ATTENUATES MECHANICAL ALLODYNIA AND CENTRAL SENSITIZATION AFTER SPINAL CORD INJURY (SCI).	Therapeutic Intervention, Model Characterization/Behavioral Measures	SCI	Claire	Hulsebosch	University of Texas Medical Branch	Hulsebosch Claire E., Crown Eric D., Gwak Young S., Johnson Kathia M., Nesci-Taylor Olivera, McAdoo David J., Perez-Polo J. Regino

243	SPASTICITY AND GAIT DISABILITIES INDUCED BY CERVICAL SPINAL CORD CONTUSION INJURY (C-SCI).	Therapeutic Intervention, Model Characterization/ Behavioral Measures	SCI	Prodip	Bose	North Florida/South Georgia VA Medical Center	Bose Prodip, Hou Jiamei, Padke Chetan, White Todd, Reier Paul, Hoffman Paul, Thompson Floyd
244	THE EFFECTS OF HYPERTONIC SALINE AND NICOTINAMIDE ON BEHAVIORAL AND COGNITIVE FUNCTION FOLLOWING CORTICAL CONTUSION INJURY	Therapeutic Intervention, Model Characterization/ Behavioral Measures	TBI	Andrea	Quigley	Southern Illinois University Carbondale	Quigley Andrea, Tan Arlene, Hoane Michael
245	THE MULTIVARIATE CONCENTRIC SQUARE FIELD TEST REVEALS DIFFERENT BEHAVIORAL PROFILES REGARDING RISK TAKING, RISK ASSESSMENT AND EXPLORATION, IN MICE SUBJECTED TO CONTROLLED CORTICAL IMPACT	Therapeutic Intervention, Model Characterization/ Behavioral Measures	TBI	Sara	Ekmark Lewén	Sweden	Ekmark Lewén Sara, Lewén Anders, Meyerson Bengt J., Hillered Lars

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POSTER SESSION D - TUESDAY 2:30 - 3:45

P#	TITLE	ASPECTS	AREA	FIRST	LAST	INSTITUTION	ALL AUTHORS
248	IN VIVO AND NON-INVASIVE ANALYSIS OF SPINAL CORD DEFORMATIONS FOR RODENT RESEARCH USING MAGNETIC RESONANCE IMAGING	Anatomy/Histology/Imaging	SCI	Kevin	Ming	University of British Columbia	Ming Kevin, Abugarbieh Rafeef, Jones Claire F., Greaves Carolyn, Yung Andrew, Kozlowski Piotr, Tetzlaff Wolfram, Crompton Peter A.
249	LONGITUDINAL VOLUMETRIC MRI STUDY OF PITUITARY GLAND FOLLOWING SEVERE TRAUMATIC BRAIN INJURY	Anatomy/Histology/Imaging	TBI	Sorin	Craciunas	KANSAS UNIVERSITY MEDICAL CENTER	Craciunas Sorin C., Cirstea Carmen M., Brooks William M.
250	ASSESSING INJURY SEVERITY AS A PREDICTOR OF BRAIN ATROPHY IN TBI	Anatomy/Histology/Imaging	TBI	William	Brooks	University of Kansas Medical Center	Brooks William, Craciunas Sorin, Yeh Hung-Wen, Lierman Joanne, Schmitt Allan, Savage Cary
251	DIFFUSE INCREASES IN CELL PROLIFERATION FOLLOWING TRAUMATIC BRAIN INJURY OF THE IMMATURE POSTNATAL DAY (PND) 17 RAT	Anatomy/Histology/Imaging	TBI	John	Caltagarene	University of Pittsburgh	Caltagarene John, Gobbel Glenn T., Srinivas Ravi, Fellows-Mayle Wendy, Adelson P. David
252	TWO-PHOTON IN VIVO IMAGING OF ASTROCYTE CALCIUM SIGNALING FOLLOWING TRAUMATIC BRAIN INJURY	Anatomy/Histology/Imaging	TBI	YungChia	Chen	University of Pennsylvania	Chen YungChia, Choo Anthony M, Miller William J, Haydon Philip G, Meaney David F
253	SUBREGIONAL MECHANICAL PROPERTIES CONTRIBUTE TO LOCAL BIOMECHANICAL RESPONSES FOLLOWING TRAUMATIC BRAIN INJURY	Anatomy/Histology/Imaging	TBI	Liyang	Zhang	Wayne State University	Zhang Liyang, Mao Haojie
254	EFFECTS OF DIFFERENT EXTRACELLULAR MATRIX MOLECULES ON THE SURVIVAL, PROLIFERATION, MIGRATION AND PROCESS EXTENSION OF OLIGODENDROCYTE PROGENITOR CELLS IN VITRO	Cellular-Molecular Signaling Pathways	In Vitro Injury	Jianguo	Hu	Stark Neurosciences Research Institute, Indiana University School of Medicine	Hu Jianguo, Deng Lingxiao, wang Xiaofei, Xu Xiao-Ming
255	VERIFICATION OF PROTEOMIC-BASED PROTEIN CHANGES BY WESTERN BLOT ANALYSIS IN A RAT MODEL OF PENETRATING BALLISTIC-LIKE BRAIN INJURY	Cellular-Molecular Signaling Pathways	TBI	Changping	Yao	Walter Reed Army Institute of Research	Yao Changping, Lu Xi-Chun May, Liao Zhilin, Torres Monika A., Wei Hans, Ottens Andrew K., Wang Kevin K., Hayes Ronald L., Tortella Frank C., Dave Jit R.
256	ATTENUATION OF BOTH NEUROINFLAMMATION AND APOPTOSIS WITH NNZ-2566 TREATMENT IN EXPERIMENTAL PBTBI.	Cellular-Molecular Signaling Pathways	TBI	Hans	Wei	Walter Reed Army Institute of Research	Wei Hans, Lu May, Phillips Katie, Yao Changping, Tortella Frank, Dave Jitendra
257	STRIATAL ADENOSINE A2A-RECEPTOR AND DOPAMINE D2-RECEPTOR HETERODIMER EXPRESSION FOLLOWING CONTROLLED CORTICAL IMPACT	Cellular-Molecular Signaling Pathways	TBI	James	Bales	University of Pittsburgh	Bales James, Yan Hong, Kochanek Patrick, Dixon C. Edward
258	NEUROGLOBIN EXPRESSION IS NOT INCREASED IN EXPERIMENTAL TRAUMATIC BRAIN INJURY	Cellular-Molecular Signaling Pathways, Anatomy/Histology/Imaging	TBI	Clay	Goodman	Baylor College of Medicine	Goodman Clay, Cherian Leela, Herron Alan, Robertson Claudia
259	TRAUMATIC BRAIN INJURY CAUSES PLASMA MEMBRANE DAMAGE AND CELL DEATH IN THE HIPPOCAMPUS OF RATS	Cellular-Molecular Signaling Pathways, Anatomy/Histology/Imaging	TBI	Chris	Lessing	Georgia Institute of Technology	Lessing M. Christian, LaPlaca Michelle
260	APOE ISOFORMS AFFECT EARLY APOPTOSIS OF NEURON AND GLIAL CELLS VIA DELAYED RECTIFYING K+ CHANNEL AFTER EXPERIMENTAL TRAUMATIC BRAIN INJURY	Cellular-Molecular Signaling Pathways, Electrophysiology	In Vitro Injury	Yong	Jiang	First Affiliated Hospital of Chongqing Medical University	Jiang Yong, Sun Xiaochuan, Feng Ming, Hu Jing, Chen Ligang, Guo Zongduo, Wu Haitao
261	REACTIVE OXYGEN SPECIES POTENTIATE NOCICEPTIVE TRANSMISSION AT BELOW REGION FOLLOWING SPINAL CORD INJURY	Cellular-Molecular Signaling Pathways, Electrophysiology	SCI	Claire	Hulsebosch	University of Texas Medical Branch	Gwak Young, Hulsebosch Claire
262	EFFECT OF DILANTIN THERAPY ON HIPPOCAMPUS CELL SURVIVAL AND REGIONAL NEUROPLASTICITY AFTER CONTROLLED CORTICAL IMPACT	Cellular-Molecular Signaling Pathways, Growth Factors/Regeneration/Plasticity	TBI	Amy	Wagner	University of Pittsburgh	Darrah Shaun, Wagner Amy K.
263	DETECTION OF MATRIX METALLOPROTEINASE-3 IN VENTRICULOSTOMY CEREBROSPINAL FLUID FOLLOWING TRAUMATIC BRAIN INJURY	Cellular-Molecular Signaling Pathways, Inflammation/Neurotoxicity	TBI	Leopold	Arko	University of New Mexico	Arko Leopold, Grossetete Mark, Phelps Jeremy, Yonas Howard, Rosenberg Gary
264	CHANGES IN AQUAPORIN CHANNEL EXPRESSION AFTER TRAUMATIC BRAIN INJURY	Cellular-Molecular Signaling Pathways, Metabolism/Physiological Assessments	TBI	Coleen	Atkins	University of Miami Miller School of Medicine	Falo M. Cristina, Sanchez-Molano Juliana, Alonso Ofelia F., Bramlett Helen M., Atkins Coleen M.
265	THE IMPORTANCE OF AQP4 PROTEIN EXPRESSION ON BRAIN EDEMA FORMATION PROCESS IN EXPERIMENTAL CORTICAL CONTUSION MODEL	Cellular-Molecular Signaling Pathways, Model Characterization/Behavioral Measures	Ischemic-Hypoxic Injury	Christina R.	Marmarou	Virginia Commonwealth University	Taya Keisuke, Marmarou Anthony, Okuno Kenji, Marmarou Christina R.

266	GROWTH OF PURIFIED ENDOGENOUS NG2+ PROGENITORS IS INHIBITED BY MICROGLIA/MACROPHAGES FROM THE INJURED SPINAL CORD	Growth Factors/ Regeneration/Plasticity	SCI	Junfang	Wu		Wu Junfang, Yoo Soonmoon, Lytle Judith, Leung Philberta, Wrathall Jean
267	TRANSPLANTATION OF EMBRYONIC STEM (ES) CELL-DERIVED OLIGODENDROCYTES ATTENUATES THE CHRONIC NEUROPATHIC PAIN CAUSED BY SPINAL CORD INJURY (SCI)	Growth Factors/ Regeneration/Plasticity	SCI	Qun	Li	Kennedy Krieger Institute	Li Qun, Tao Feng, Liu Su, Johns Roger A., McDonald John W.
268	PRO-APOPTOTIC FUNCTION FOR EPH RECEPTORS DURING ADULT NEUROGENESIS AND FOLLOWING CNS INJURY	Growth Factors/ Regeneration/Plasticity	TBI	Michelle	Hendrick-Theus	University of Miami	Hendrick-Theus Michelle, Ricard Jerome, Liebl Daniel
269	CONDITIONAL KNOCKOUT OF BRAIN-DERIVED NEUROTROPHIC FACTOR IN THE HIPPOCAMPUS INCREASES DEATH OF ADULT-BORN IMMATURE NEURONS FOLLOWING TRAUMATIC BRAIN INJURY	Growth Factors/ Regeneration/Plasticity	TBI	Jinhui	Chen		Gao Xiang, Chen Jinhui
270	EFFECT OF TRAUMATIC BRAIN INJURY ON THE NORMALLY QUIESCENT POPULATION OF NEURAL STEM CELLS IN THE ADULT MOUSE SVZ.	Growth Factors/ Regeneration/Plasticity	TBI	Neil	Harris	UCLA	Miller Gretchen, Le Belle Janel, Hovda David, Kornblum Harley, Harris Neil
271	MORPHOLOGICAL ANALYSIS OF DISSOCIATED DORSAL ROOT GANGLION NEURONS FOLLOWING SPINAL CORD INJURY	Growth Factors/ Regeneration/Plasticity, Anatomy/Histology/Imaging	SCI	Supinder	Bedi	University of Texas, Medical School at Houston	Bedi Supinder, Lago Michael, Grill Raymond, Walters E.T.
273	EFFECTS OF BFGF INCORPORATED GELATIN HYDROGEL TRANSPLANTATION IN A RAT SPINAL CORD CONTUSION MODEL	Growth Factors/ Regeneration/Plasticity, Model Characterization/ Behavioral Measures	SCI	Takeo	Furuya	Chiba University	Furuya Takeo, Hashimoto Masayuki, Koda Masao, Murata Atsushi, Okawa Akihiko, Yamazaki Masashi, Dezawa Mari, Matsuse Dai, Tabata Yasuhiko, Takahashi Kazuhisa
274	ALBUMIN ACTIVATES ASTROCYTES VIA ACTIVATION OF MAPKS AND THE TGF β -RECEPTOR	Inflammation/Neurotoxicity	In Vitro Injury	Hanta	Ralay-Ranaivo	Northwestern University Feinberg School of Medicine	Ralay-Ranaivo Hanta, Wainwright Mark
275	AMPA PEPTIDE ASSAY FOR CONCUSSIONS AND MILD TRAUMATIC BRAIN INJURY.	Inflammation/Neurotoxicity	TBI	Svetlana	Dambinova	Emory University	Dambinova Svetlana, Tcherepanov Andrew, Shumilina Maria, Khunteev German, Izykenova Galina
276	SINGLE DOSE OF NEUROTOXIN MPTP PERMANENTLY ALTERS MITOCHONDRIAL BIOENERGETICS IN THE RHESUS MONKEY NIGROSTRIATAL SYSTEM	Inflammation/Neurotoxicity	TBI	Jignesh	Pandya	SCoBIRC	Pandya Jignesh, Grondin Richard, Zhang Ziming, Cass Wayne, Chen Anshu, Gerhardt Greg, Springer Joe, Gash Don, Sullivan Patrick
277	ALTERATIONS IN BLOOD-BRAIN BARRIER PERMEABILITY TO LARGE AND SMALL MOLECULES AND LEUKOCYTE ACCUMULATION AFTER TRAUMATIC BRAIN INJURY: EFFECTS OF THERAPEUTIC HYPOTHERMIA	Inflammation/Neurotoxicity, Anatomy/Histology/Imaging	TBI	George	Lotocki	University of Miami Miller School of Medicine	Lotocki George, De Rivero Vaccari Juan Pablo, Perez Enrique, Sanchez-Molina Juliana, Furones-Alonso Ofelia, Bramlett Helen M., Dietrich W. Dalton
278	EVIDENCE FOR FAS MEDIATED APOPTOSIS AND INFLAMMATION IN THE PATHOMECHANISMS OF HUMAN CERVICAL SPONDYLOTIC MYELOPATHY-A FORM OF NON-TRAUMATIC SPINAL CORD INJURY	Metabolism/Physiological Assessments	SCI	Wenru	Yu	Toronto Western Hospital Research Institute, Krembil Neuroscience Center, University of Toronto	Yu Wenru, Fehlings Michael G
279	MICRODIALYSIS AS A POSSIBLE PREDICTOR OF SIGNIFICANT SECONDARY INJURY IN PATIENTS WITH SEVERE TRAUMATIC BRAIN INJURY.	Metabolism/Physiological Assessments	TBI	Alexander	Tuchman	University of Miami Miller School of Medicine	Tuchman Alexander, Bullock Ross
281	REGIONAL CEREBRAL BLOOD FLOW RESPONSE FOLLOWING BRAIN INJURY DEPENDS ON DIRECTION OF HEAD MOTION	Metabolism/Physiological Assessments	TBI	Stephanie	Eucker	University of Pennsylvania	Eucker Stephanie, Friess Stuart, Ralston Jill, Margulies Susan
282	STUDIES OF BLOOD AND CSF BIOMARKERS OF CNS DAMAGE AND DISEASE	Model Characterization/ Behavioral Measures	Ischemic-Hypoxic Injury	Gerry	Shaw	University of Florida	Shaw Gerry, Lewis Stephen, Howland Dena, Anderson Kevin, Weiss Michael, Yang Cui, Borchelt David
283	ASSESSMENT OF AGE AND COMORBIDITY INDEXES AS POTENTIAL PREDICTORS OF CLINICAL OUTCOMES IN PATIENTS WITH ACUTE SPINE TRAUMA	Model Characterization/ Behavioral Measures	SCI	Julio	Furlan	Toronto Western Research Institute	Furlan Julio, Kattail Deepa, Fehlings Michael
284	QUANTITATIVE ASSAYS OF PROTEOLYTIC DEGRADATION OF A-SPECTRIN IN HUMAN CSF FOLLOWING SEVERE TRAUMATIC BRAIN INJURY (TBI)	Model Characterization/ Behavioral Measures	TBI	Andras	Buki	Banyan Biomarkers Inc	Robinson Gillian, Buki Andras, Gabrielli Andrea, Robicsek Steven, Tepas Joseph, Pineda Jose, Robertson Claudia, Oli Monika, Akinyi Linnet, Mo Jixiang, Scharf Dancia, Liu Ming Chen, Zheng Wenrong, Wang Kevin, Hayes Ronald

285	VALIDITY OF THE NEUROPHYSICAL OUTCOME SCALE IN TRAUMATIC BRAIN INJURY AND FEASIBILITY FOR USE IN CLINICAL RESEARCH	Model Characterization/ Behavioral Measures	TBI	Elisabeth	Wilde	Baylor College of Medicine	Wilde Elisabeth, McCauley Stephen, Kelly Tara, Weyand Annie, Levin Harvey, Clifton Guy, Valadka Alex, Boake Corwin, Shah Monika, Robertson Claudia, Moretti Paolo
286	NOVEL MODEL OF BLAST OVERPRESSURE BRAIN INJURY: A COMPREHENSIVE PLATFORM FOR PROFILING MOLECULAR & CELLULAR MECHANISMS AND SYSTEMS BIOLOGY STUDIES	Model Characterization/ Behavioral Measures	TBI	Stanislav	Svetlov	Banyan Biomarkers, Inc.	Svetlov Stanislav, Prima Victor, Lerner Stephen, Tang Qiushi, Atkinson Joseph, Gutierrez Hector, Kirk Daniel, Hayes Ronald, Wang Kevin
287	VISUALIZATION OF CHANGES IN SODIUM CHANNEL SEQUESTRATION IN AXONAL INJURY FOLLOWING DIFFUSE TRAUMATIC BRAIN INJURY	Model Characterization/ Behavioral Measures	TBI	Christina R.	Marmarou	Virginia Commonwealth University	Marmarou Christina R, Taya Keisuke, Liang Xiuyin, Abidi Nageeb, Pomciter Anthony, Dupree Jeff, Marmarou Anthony
288	DETECTION OF A PERSISTENT BEHAVIORAL SENSORY MORBIDITY AFTER TBI TO EVALUATE REHABILITATIVE INTERVENTIONS IN THE RAT	Model Characterization/ Behavioral Measures	TBI	Katelyn	McNamara	University of Kentucky	McNamara Katelyn, Lisembee Amanda, Lifshitz Jonathan
289	CATWALK GAIT ANALYSIS FOR THE ASSESMENT OF BEHAVIORAL DEFICITS FOLLOWING TRAUMATIC BRAIN INJURY	Model Characterization/ Behavioral Measures	TBI	Shan	Sharif	Georgia Institute of Technology	Sharif Shan, Simon Crystal, Lessing M. Christian, LaPlaca Michelle
291	DIFFUSE AXONAL INJURY IN RAT MODEL BY LATERAL HEAD ROTATION MORPHOLOGICAL STUDY	Model Characterization/ Behavioral Measures, Anatomy/Histology/Imaging	TBI	Xiaosheng	He	Xijing Hospital, Fourth Military Medical University	He Xiaosheng, Zhang Xiang, Fei Zhou
292	HISTOLOGICAL AND BEHAVIORAL EFFECTS OF CALPASTATIN OVEREXPRESSION AFTER TRAUMATIC BRAIN INJURY IN TRANSGENIC MICE	Model Characterization/ Behavioral Measures, Anatomy/Histology/Imaging	TBI	Heather	Foozer	University of Kentucky	Foozer Heather, Pleasant Jennifer, Schoch Kathleen, Saatman Kathryn
293	CHANGES IN EXTRACELLULAR POTASSIUM IFLUENCE INJURY-RELATED NEURONAL ACTIVITY AND CELL DEATH	Model Characterization/ Behavioral Measures, Inflammation/Neurotoxicity	In Vitro Injury	Gene	Gurkoff	University of California Davis	Gurkoff Gene, Katleba Kimberley, Beller Justin, Lyeth Bruce
294	CALPAIN MEDIATED PROTEOLYSIS OF TAU IN RAT BRAIN AFTER TRAUMATIC BRAIN INJURY	Model Characterization/ Behavioral Measures, Inflammation/Neurotoxicity	TBI	Ming Cheng	Liu	Banyan Biomarkers Inc.	Liu Ming Cheng, Zheng Wenrong, Kobeissy Firas H., Hayes Ronald L., Wang Kevin K.W.
295	CEREBROSPINAL FLUID PRESSURES ASSOCIATED WITH SPINAL CORD INJURY: PILOT STUDY IN A LARGE ANIMAL MODEL	Model Characterization/ Behavioral Measures, Metabolism/Physiological Assessments	SCI	Claire	Jones	University of British Columbia	Jones Claire F., Kwon Brian K., Crompton Peter A.
296	EXAMINATION OF THE EARLY EFFECTS OF NEURAL PRECURSOR CELL TRANSPLANTATION ON THE PERI-LESIONAL ENVIRONMENT OF THE INJURED SPINAL CORD.	Therapeutic Intervention	SCI	Gregory	Hawryluk	University of Toronto	Hawryluk Gregory, Karmimi- Abdolrezaee Soheila, Eftekharpour Eftekhar, Fehlings Michael
297	TARGETING PHOSPHODIESTERASE-4 AFTER SPINAL CORD INJURY USING PHARMACOLOGICAL AND MOLECULAR APPROACHES	Therapeutic Intervention	SCI	Damien	Pearse		Schaal Sandra M, Garg Maneesh S, Golshani Roozbeh, Ghosh Mousumi, Lovera Lillie, Lopez Michael, Patel Monal, Louro Jack, Puentes Rocio, Sanchez Andre R, Andrade Christian M, Tuesta Luis M, Diaz Paulo, Iorgulescu Bryan, Puzis Leopold, Pearse Damien D
298	FUNCTIONAL EFFECTIVENESS OF OLFACTORY MUCOSA COMPARED TO PROGENITOR CELLS DERIVED FROM OLFACTORY MUCOSA OR BONE MARROW IN OUTBRED AND INBRED STRAINS OF RATS WITH CHRONIC, SEVERE SPINAL CORD INJURY	Therapeutic Intervention	SCI	Jean	Peduzzi	Wayne State University School of Medicine	Peduzzi Jean, Lima Carlos, Meythaler Jay
299	DISPROPORTIONATE DEFICITS IN EXECUTIVE FUNCTIONING, MEMORY AND JUDGMENT FOLLOWING TBI: EVIDENCE FROM THE LEBBY-ASBELL NEUROCOGNITIVE SCREENING EXAMINATION FOR CHILDREN (LANSE-C) AND ADOLESCENTS (LANSE-A).	Therapeutic Intervention	TBI	Shana	Asbell	Mary Free Bed Hospital	Lebby Paul, Asbell Shana, Kennington Neal, Johnson William, Jackson Sarah, Andrichuk Lyudmila, Turitz Michelle, Chapman-Cutley Marilyn, Gonzalez Natalie
300	MEASUREMENT OF BRAIN OXYGENATION(PBTO2) AS PART OF A MULTIMODALITY MONITORING APPROACH TO THE MANAGEMENT OF CHILDREN WITH SEVERE TRAUMATIC BRAIN INJURY	Therapeutic Intervention	TBI	Martina	Stippler	University of Pittsburgh	Stippler Martina, Adelson David, Brown Daniela, Chang Yuefang, Grandhi Ramesh, Bell Michael

301	A WEB-BASED INTEGRATED MEDICAL INFORMATION SYSTEM FOR SEVERE TRAUMATIC BRAIN INJURY	Therapeutic Intervention	TBI	Beng-Ti	Ang	National Neuroscience Institute, Singapore	Ang Beng-Ti, Zhang Zhuo, Tang Sui Sheng, Lim Tchoyoson, Tan Chew-Lim, Gong Tian-Xia, Liu Rui-Zhe, Tian Qi
302	POSTINJURY MEMORY MODULATION BY GLUCOSE IS DEPENDENT ON TIME OF ADMINISTRATION	Therapeutic Intervention	TBI	Olga	Kokiko-Cochran	Virginia Commonwealth University	Kokiko-Cochran Olga, Hamm Robert
304	THE CEREBROVASCULAR PROTECTIVE EFFECTS OF HYPOTHERMIA AFTER TRAUMATIC BRAIN INJURY COMPLICATED BY A SECONDARY INSULT IN RATS.	Therapeutic Intervention	TBI	Guoyi	Gao		Gao Guoyi, Wei Enoch, Povlishock John
305	THE NITRONE FREE RADICAL SCAVENGER NXY-059 IS NEUROPROTECTIVE WHEN ADMINISTERED AFTER TRAUMATIC BRAIN INJURY IN THE RAT	Therapeutic Intervention	TBI	Fredrik	Clausen	Uppsala University	Clausen Fredrik, Marklund Niklas, Lewén Anders, Hillered Lars
306	CONTROLLED NEGATIVE PRESSURE REDUCES BRAIN EDEMA AND MODULATES BRAIN METABOLITES IN A BRAIN INJURY MODEL	Therapeutic Intervention	TBI	Zhenlin	Zheng	Wake Forest University Health Science	Argenta Louis, Morykwas Michael, Zheng Zhen-lin, Wagner William, Tatter Stephen
307	HUMAN AMNION-DERIVED MULTIPOTENT PROGENITOR (AMP) CELLS SEATED IN A COLLAGEN SCAFFOLD PROMOTE WOUND RECOVERY: PRE-CLINICAL STUDIES IN AN EXPERIMENTAL MODEL OF PBBi.	Therapeutic Intervention, Anatomy/Histology/Imaging	TBI	Zhiyong	Chen	Walter Reed Army Institutes of Research	Chen Zhiyong, Evangelista Clifford, Lu X.-C. May, Clarke Diana, Sing George, Tortella Frank
308	A NEW LARGE ANIMAL MODEL OF TRAUMATIC BRAIN INJURY	Therapeutic Intervention, Anatomy/Histology/Imaging	TBI	Sandya	Venugopal	SFVAH	Venugopal Sandya, Coppes Valerie, Moody Erin, Panter Scott
309	SODIUM CHANNELOPATHY: A POTENTIAL MECHANISM CAUSING EXACERBATED DAMAGE WITH REPETITIVE MILD TRAUMATIC AXON INJURY.	Therapeutic Intervention, Cellular-Molecular Signaling Pathways	In Vitro Injury	Tracy	Yuen	University of Pennsylvania	Yuen Tracy, Browne Kevin, Iwata Akira, Smith Douglas
310	THE EFFECT OF OXYGENATION LEVEL ON CEREBRAL POST-TRAUMATIC APOPTOSIS IS MODULATED BY THE 18 KDA TRANSLOCATOR PROTEIN IN A RAT MODEL OF CORTICAL CONTUSION	Therapeutic Intervention, Cellular-Molecular Signaling Pathways	TBI	Jean	Soustiel	Faculty of Medicine, Technion Israel Institute of Technology	Soustiel Jean Francois, Veenman Leo, Zaaroor Menashe, Gavish Moshe
311	A LAMININ-BASED SCAFFOLD FOR IMPROVED NEURAL STEM CELL SURVIVAL	Therapeutic Intervention, Cellular-Molecular Signaling Pathways	TBI	Michelle	LaPlaca	Georgia Institute of Technology	Stabenfeldt Sarah, García Andrés, LaPlaca Michelle
312	BIOLOGICAL PROPERTIES OF HYALURONIC ACID IN GLIAL SCAR FORMATION AFTER SCI	Therapeutic Intervention, Cellular-Molecular Signaling Pathways, Inflammation/Neurotoxicity	SCI	Zin	Khaing	University of Texas at Austin	Khaing Zin, Seidlits Stephanie, Grill Raymond, Schmidt Christine
313	CAUDA EQUINA INJURY RESULTING IN BLADDER-SPHINCTERIC FUNCTIONAL DEFICITS AND PERMANENT LOSS OF DORSAL HORN AFFERENTATION	Therapeutic Intervention, Growth Factors/Regeneration/Plasticity	SCI	Brian	Rooney	University of Texas, Medical Branch	Rooney Brian, Hulsebosch Claire
314	S100B ENHANCES HIPPOCAMPAL PROGENITOR CELL PROLIFERATION AND SURVIVAL FOLLOWING AN UNILATERAL PARIETAL BRAIN LESION	Therapeutic Intervention, Growth Factors/Regeneration/Plasticity	TBI	Andrea	Kleindienst	University Erlangen-Nuremberg/ Klinikum Amberg	Kleindienst Andrea, Gruenbeck Felicitas, Emtmann Irene, Buslei Rolf, Koefel Katharina, Eck Kattarina, Buchfelder Michael
315	POST-INJURY ADMINISTRATION OF ATOMOXETINE ENHANCES NEUROPLASTICITY IN THE HIPPOCAMPUS FOLLOWING LATERAL FLUID-PERCUSSION INJURY	Therapeutic Intervention, Growth Factors/Regeneration/Plasticity	TBI	Wendy	Reid	Virginia Commonwealth University	Reid Wendy, Hamm Robert J.
316	EVALUATION OF THE THERAPEUTIC POTENTIAL OF AN INJECTABLE BLEND OF HYALURONAN (HA) AND METHYL CELLULOSE (MC) FOLLOWING EXPERIMENTAL SPINAL CORD INJURY	Therapeutic Intervention, Inflammation/Neurotoxicity	SCI	James	Austin	University of Toronto Western Research Institute	Austin James, Kang Catherine, Baumann Douglas, Shoichet Molly, Fehlings Michael
317	HDAC INHIBITOR INCREASES HISTONE H3 ACETYLATION AND REDUCES MICROGLIA INFLAMMATORY RESPONSE FOLLOWING TRAUMATIC BRAIN INJURY IN RATS	Therapeutic Intervention, Inflammation/Neurotoxicity	TBI	Bin	Zhang	UC Davis	Zhang Bin, West Eric, Van Ken, Gurkoff Gene, Kozikowski Alan, Lyeth Bruce
318	TRAUMATIC BRAIN INJURY-INDUCED EDEMA IN SUB-CORTICAL STRUCTURES IS REVERSED BY ACUTE PROGESTERONE TREATMENT IN AGED OVARECTOMIZED RATS.	Therapeutic Intervention, Inflammation/Neurotoxicity	TBI	Badrinarayana	Kasturi n	Emory University School of Medicine	Kasturi Badrinarayanan, Kemkar Sneha, Gandhi Priya, Stein Donald
319	HYPOPIUITARISM ASSOCIATED WITH TRAUMATIC BRAIN INJURY – ANALYSIS OF THE PÉCS TRAUMATIC BRAIN INJURY DATABASE	Therapeutic Intervention, Metabolism/Physiological Assessments	TBI	Dora	Szellar	Pécs University, Hungary	Szellar Dora, Mezosi Emese, Nemes Orsolya, Nagy Zsuzsanna, Bodis Beata, Bajnok Laszlo, Czeiter Endre, Doczi Tamas, Buki Andras

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The Women in Neurotrauma Research Visiting International Scholar Award (WINTR-VISA) was established to promote international networking opportunities, and to advance the careers of young neurotrauma researchers. The Award provides an opportunity for international collaboration by subsidizing both travel to the National Neurotrauma Symposium and a brief period of research training/collaboration in a sponsor's laboratory. This award supports the mission of WINTR **to promote international gender equality in neurotrauma research.**

The WINTR-VISA provides full financial support for a woman neurotrauma researcher (doctoral student, post-doctoral fellow, or junior independent scientist) to attend the 26th Annual National Neurotrauma Symposium. In addition, the award subsidizes a research training/collaborative opportunity in any neurotrauma laboratory in the United States to learn new skills or techniques pertinent to their research project.

The WINTR-VISA Committee is pleased to announce **Ms. Ashley Di Battista** as the 2008 WINTR Visiting International Scholar. The committee unanimously selected her application, because it reflects the purpose of the award. Ashley is studying Brain and Behavioral Psychology at York University and is conducting brain injury research at The Hospital for Sick Children in Toronto, Canada. Currently, she is working with Dr. Anne-Marie Guerguerian on clinical pediatric TBI research, and their current project is entitled "*Systematic Review of Acute Phase Observational Studies Measuring Neuropsychological and Functional Outcomes in Children following Severe Traumatic Brain injury.*" In Dr. Christopher Giza's laboratory at UCLA, she will have the opportunity to learn how her clinical work can be translated into the framework of a Neurotrauma basic science program. The experience will also expose her to the management of neurotrauma in the United States. Through this award, WINTR will help advance her career as a promising young neurotrauma researcher. Congratulations on your achievement! WINTR is honored to have you advance our mission.



Requests for applications will be announced in January for the 2009 WINTR-VISA. For more information, please contact the executive committee at WINTR-VISA@neurotrauma.org.



This image shows a single sheet of white paper with horizontal ruling lines. The lines are evenly spaced and run across the width of the page. There are no margins, text, or other markings on the paper.

NNS SOCIETY MEMBERSHIP

INFORMATION

There is no official deadline for membership; we welcome new members at any time!

Should you decide to become a member mid-year with the option of receiving a subscription to Journal of Neurotrauma, you will receive the back issues starting January of the same year of your membership.

Membership with Journal Subscription includes ELECTRONIC ACCESS to the Journal of Neurotrauma (LIEBERT ONLINE).

The benefits include:

- *Complete searching capabilities of full text, abstracts, TOC, figures and references.*
- *Citations & references linked to external bibliographic resources on the Web (e.g. MEDLINE).*
- *Cross-title, cross-publisher linking on the article-to-article level via CrossRef (www.crossref.org).*
- *Dependable Access, 24 hours a day, 7 days a week, ensured by a global network of dedicated servers.*
- *TOC email alert each time a new issue is online.*
- *Overseas members get to see new issues more quickly, since online is up before print copy arrives.*
- *Easy password validation and gateway to the online version.*

To apply, complete the Application form on the reverse side of this page
and mail it along with your Curriculum Vitae and payment to:

Linda Garcia
National Neurotrauma Society
8032 SW 45 Lane
Gainesville FL 32608 USA

If you would like to be added to our mailing list, please contact the National Office:

TEL: (352) 271-1169
FAX: (305) 704-3814
E-MAIL: garcialinda22@gmail.com

APPLICATION

Please Print

Name (Last, First, MI) _____ M.D. _____ Ph.D. _____ Other _____

Position/Title _____

Department/Institution _____

Mailing Address _____

City/State/Zip _____

Office Phone _____ Fax _____ E-mail (please print clearly) @ _____

Research Interests _____

MEMBERSHIP CATEGORIES

Enclose curriculum vitae and check (US bank only) or credit card info. Check one:

- ☐ Membership Jan.-Dec. 2009 with 12 monthly issues of the *Journal of Neurotrauma* (\$245.00)
- ☐ Student, Fellow, Resident membership Jan.-Dec. 2009 with 12 monthly issues of the *Journal of Neurotrauma* (\$195.00)
- ☐ Membership Jan.-Dec. 2009 without subscription to the *Journal of Neurotrauma* (\$100.00)
- ☐ Student, Fellow, Resident membership Jan.-Dec. 2009 without subscription to the *Journal of Neurotrauma* (\$50.00)

MEMBERSHIP TO WOMEN IN NEUROTRAUMA (WINTR) (elective)

- ☐ Regular membership to WINTR for Jan.-Dec. 2009 (\$30.00)
- ☐ Student/post-doc membership to WINTR for Jan.-Dec. 2009 (\$10.00)

TOTAL AMOUNT PAID (U.S. Dollars only) \$ _____

Checks or money orders (US funds only) should be made payable to the **National Neurotrauma Society** (Tax I.D. #76-0520736).

If paying by credit card, provide the following info:

____ MasterCard ____ Visa ____ American Express

Card Number _____

Expiration Date __ / __ / __ (mm/dd/yy) Zip code of cardholder (U.S. only) _____

Cardholder Name (print) _____

Cardholder Signature _____

PROGRAM EVALUATION FORM

Please rate each speaker presentation by circling the appropriate letter in both columns.
(A=Excellent, B=Good, C=Fair, D=Poor)

ARE YOU AN:

☐ MD ☐ PhD ☐ Other

SPEAKER	CONTENT	PRESENTATION	SPEAKER	CONTENT	PRESENTATION
Special Session on Sunday 7/27			WINTR Special Session		
Bullock	A B C D	A B C D	Hicks	A B C D	A B C D
Adelson	A B C D	A B C D	Session 7A		
Maas	A B C D	A B C D	Noble-Haesslein	A B C D	A B C D
Session 1			Bayir	A B C D	A B C D
Houle	A B C D	A B C D	Hall	A B C D	A B C D
Zheng	A B C D	A B C D	Session 7B		
G. Smith	A B C D	A B C D	Maeda	A B C D	A B C D
Session 3A			Aarabi	A B C D	A B C D
Raghupathi	A B C D	A B C D	Ecklund	A B C D	A B C D
Courtney Robertson	A B C D	A B C D	Session 8A		
Prins	A B C D	A B C D	Faden	A B C D	A B C D
Session 3B			Kocsis	A B C D	A B C D
Green	A B C D	A B C D	Sofroniew	A B C D	A B C D
Kwon	A B C D	A B C D	Session 8B		
Fehlings	A B C D	A B C D	Claudia Robertson	A B C D	A B C D
Session 4A			Manley	A B C D	A B C D
Ling	A B C D	A B C D	Vespa	A B C D	A B C D
Kochanek	A B C D	A B C D	Session 9		
Cernak	A B C D	A B C D	Lovell	A B C D	A B C D
Session 4B			D. Smith	A B C D	A B C D
Fehlings	A B C D	A B C D	Hoge	A B C D	A B C D
Okonkwo	A B C D	A B C D	Session 10		
Session 5			Manley	A B C D	A B C D
Clark	A B C D	A B C D	Hicks	A B C D	A B C D
Tetzlaff	A B C D	A B C D			
Hovda	A B C D	A B C D			

In your opinion, was information presented by each speaker presented fairly and without commercial or promotional bias?

☐ YES ☐ NO If no, please explain: _____

Please rate the overall program by circling the appropriate letter in both columns. (A=Excellent, B=Good, C=Fair, D=Poor)

Dates of meeting	A B C D	City Location	A B C D
Educational content	A B C D	Hotel Selection	A B C D
Open Communications Sessions	A B C D	General Sessions	A B C D
Poster Abstract Presentations	A B C D	Student Networking Happy Hour	A B C D
WINTR Reception	A B C D	Welcome Reception	A B C D
WINTR Lunch Session	A B C D	NNS Business Meeting	A B C D
Networking Opportunities	A B C D	Website Information	A B C D
Website Registration	A B C D	Website Abstract Submissions	A B C D
Onsite Registration & Staff	A B C D	Organizer Communications	A B C D
"All Under One Roof" Convenience	A B C D	Overall Value	A B C D

COURSE OBJECTIVES

At the completion of this symposium, participants should be able to:

1. Describe current concepts that guide clinical care of brain and spinal cord injury patients and the therapeutic targets for improved functional recovery in the future.
2. Describe mechanisms that contribute to brain cell injury and death, and how age influences these processes after traumatic central nervous system (CNS) injury.
3. Describe interventions and therapeutic strategies that stimulate regeneration and improve outcome after CNS trauma and how they might interact with endogenous recovery mechanisms.
4. Describe how laboratory as well as clinical research is impacting patients with traumatic brain and spinal cord injury.

Did this program succeed in meeting its COURSE OBJECTIVES? ☐ YES ☐ NO If no, please explain: _____

EDUCATIONAL GOALS

The goals of the 2008 Symposium are:

1. To provide a forum for the *presentation, discussion and feedback* regarding the most recent findings in neurotrauma research and to encourage the interaction between both those new in the field and those with extensive experience in this area.
2. To present and discuss the findings of established neuroscientists engaged in other areas of research in order to encourage the *participation in neurotrauma research* of these experts as well as to provide the neurotrauma community the opportunity to incorporate these new ideas and concepts into their research.
3. To present and discuss the state of the art clinical management of both brain and spine/ spinal cord injury to clinicians and neuroscientists in order to encourage collaboration, communication, and participation in neurotrauma research and translate those concepts into clinical trials and clinical practice to improve outcomes in patients with neural injuries.
4. To provide hands on sessions for clinicians in the areas of brain and spine/ spinal cord injury to encourage the incorporation of the state of the art clinical techniques in reconstruction and multimodality monitoring in the care of the injured patient.
5. Describe the state of the art interventions, therapeutic strategies, and multimodality monitoring can be used to improve outcome after CNS trauma.
6. Describe how acute surgical intervention and later reconstruction can be utilized to improve the outcome of patients with traumatic brain and spine/ spinal cord injury.

Did this program succeed in meeting its EDUCATIONAL GOALS? ☐ YES ☐ NO If no, please explain:

NEEDS ASSESSMENT SURVEY FOR FUTURE PROGRAMS

Please list two major areas of **KNOWLEDGE OR SKILLS** that you feel must be addressed in the next few years:

a) _____ b) _____

Please give us any suggestions you have for **TOPICS** to be presented at next year's conference:

a) _____ b) _____

Please **RANK** the deciding factors in attending **NNS 2008**: (1 = most important, 4 = least important)

____ Topics ____ Location ____ Faculty ____ Time of Year ____ Other: _____

What hotel features **MOST** influence your decision to stay at the official host hotel? (Please check all that apply)

☐ Pool ☐ Fitness Center ☐ Spa ☐ Golf ☐ Tennis ☐ Kid's Programs
☐ Nightlife ☐ Spouse Tours ☐ Shopping ☐ None ☐ Other: _____

Which of the following future events do you anticipate attending? (Please check all that apply)

☐ NNS 2009 in Santa Barbara, CA ☐ 2009 SfN Annual Meeting ☐ Other: _____

Which of the following would you **MOST** like to see incorporated into the next symposium program?

☐ Additional General Session Topics ☐ Breakout Sessions ☐ Additional Poster Sessions
☐ Additional Oral Abstract Presentations ☐ Workshops ☐ Additional Networking Opportunities
☐ Other : _____

Any additional suggestions for future meetings? (Please be sure to write legibly!)

**PLEASE RETURN YOUR COMPLETED EVALUATION FORM
TO THE NNS REGISTRATION DESK. THANK YOU.**

You may also complete this survey online at: www.neurotrauma.org/2008
An email link will be sent out after the conference to all attendees.

APPENDIX II- Evaluation Results for 26th National Neurotrauma Symposium

Constant Contact Survey Results

Survey Name: NNS08 Post-Conf Survey

Response Status: Partial & Completed




Filter: None

Aug 21, 2008 9:58:58 AM

* I am a:

Panel A:			Number of Response(s)	Response Ratio
Answer	0%	100%		
MD			71	29.9 %
PhD			90	37.9 %
MD, PhD			17	7.1 %
Other			57	24.0 %
No Response(s)			2	<1 %
Totals			237	100%

* In your opinion, was the information presented by each speaker presented fairly and without commercial or promotional bias?

Answer	0%	100%	Number of Response(s)	Response Ratio
Yes			229	96.6 %
No			4	1.6 %
No Response(s)			4	1.6 %
Totals			237	100%

COURSE OBJECTIVES

At the completion of this symposium, participants should be able to:

1. Describe current concepts that guide clinical care of brain and spinal cord injury patients and the therapeutic targets for improved functional recovery in the future.
- * 2. Describe mechanisms that contribute to brain cell injury and death, and how age influences these processes after traumatic central nervous system (CNS) injury.
3. Describe interventions and therapeutic strategies that stimulate regeneration and improve outcome after CNS trauma and how they might interact with endogenous recovery mechanisms.
4. Describe how laboratory as well as clinical research is impacting patients with traumatic brain and spinal cord injury.

DID THIS PROGRAM SUCCEED IN MEETING ITS EDUCATIONAL OBJECTIVES?

Answer	0%	100%	Number of Response(s)	Response Ratio
Yes			224	94.5 %
No			9	3.7 %
No Response(s)			4	1.6 %
Totals			237	100%

Please rate the following speaker presentations for Special Session on Sunday 7/27:




1 = Excellent, 2 = Good, 3 = Fair, 4 = Poor

Answer	1	2	3	4	Number of Response(s)	Rating Score*
Ross Bullock					111	1.5
P.David Adelson					112	1.5
Andrew Maas					107	1.6

*The Rating Score is the weighted average calculated by dividing the sum of all weighted ratings by the number of total responses.

Please rate the following speaker presentations for Session 1:




1 = Excellent, 2 = Good, 3 = Fair, 4 = Poor

Answer	1	2	3	4	Number of Response(s)	Rating Score*
John Houle					135	1.6
Binhai Zheng					136	1.8
George M. Smith					134	1.6

*The Rating Score is the weighted average calculated by dividing the sum of all weighted ratings by the number of total responses.

Please rate the following speaker presentations for Session 3A:




1 = Excellent, 2 = Good, 3 = Fair, 4 = Poor

Answer	1	2	3	4	Number of Response(s)	Rating Score*
Ramesh Raghupathi					113	1.7
Courtney Robertson					111	1.8
Mayumi Prins					112	1.7

*The Rating Score is the weighted average calculated by dividing the sum of all weighted ratings by the number of total responses.

Please rate the following speaker presentations for Session 3B:




1 = Excellent, 2 = Good, 3 = Fair, 4 = Poor

Answer	1	2	3	4	Number of Response(s)	Rating Score*
Barth Green					100	1.7
Brian Kwon					103	1.5
Michael Fehlings					104	1.5

*The Rating Score is the weighted average calculated by dividing the sum of all weighted ratings by the number of total responses.

Please rate the following speaker presentations for Session 4A:




1 = Excellent, 2 = Good, 3 = Fair, 4 = Poor

Answer	1	2	3	4	Number of Response(s)	Rating Score*
Geoffrey Ling					137	1.5
Patrick Kochanek					141	1.5
Ibolja Cernak					138	1.9

*The Rating Score is the weighted average calculated by dividing the sum of all weighted ratings by the number of total responses.

Please rate the following speaker presentations for Session 4B:




1 = Excellent, 2 = Good, 3 = Fair, 4 = Poor

Answer	1	2	3	4	Number of Response(s)	Rating Score*
Michael Fehlings					66	1.5
David Okonkwo					57	1.6
Table Instructors					53	1.8

*The Rating Score is the weighted average calculated by dividing the sum of all weighted ratings by the number of total responses.

Please rate the following speaker presentations for Session 5:

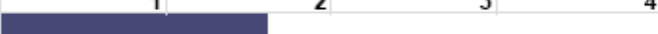
1 = Excellent, 2 = Good, 3 = Fair, 4 = Poor

Answer	1	2	3	4	Number of Response(s)	Rating Score*
Robert S.B. Clark					135	1.5
Wolfram Tetzlaff					135	1.7
David Hovda					144	1.4

*The Rating Score is the weighted average calculated by dividing the sum of all weighted ratings by the number of total responses.

Please rate the following speaker presentations for WINTR Special Session:




1 = Excellent, 2 = Good, 3 = Fair, 4 = Poor

Answer	1	2	3	4	Number of Response(s)	Rating Score*
Ramona Hicks					95	1.6

*The Rating Score is the weighted average calculated by dividing the sum of all weighted ratings by the number of total responses.

Please rate the following speaker presentations for for Session 7A:




1 = Excellent, 2 = Good, 3 = Fair, 4 = Poor

Answer	1	2	3	4	Number of Response(s)	Rating Score*
Linda Noble-Haeusslein					112	1.7
Hulya Bayir					116	1.5
Edward Hall					118	1.4

*The Rating Score is the weighted average calculated by dividing the sum of all weighted ratings by the number of total responses.

Please rate the following speaker presentations for for Session 7B:




1 = Excellent, 2 = Good, 3 = Fair, 4 = Poor

Answer	1	2	3	4	Number of Response(s)	Rating Score*
Takeshi Maeda					77	2.0
Bizhan Aarabi					76	1.9
James Ecklund					74	1.6

*The Rating Score is the weighted average calculated by dividing the sum of all weighted ratings by the number of total responses.

Please rate the following speaker presentations for for Session 8A:




1 = Excellent, 2 = Good, 3 = Fair, 4 = Poor

Answer	1	2	3	4	Number of Response(s)	Rating Score*
Alan Faden					96	1.7
Jeffrey Kocsis					93	1.6
Michael Sofroniew					94	1.5

*The Rating Score is the weighted average calculated by dividing the sum of all weighted ratings by the number of total responses.

Please rate the following speaker presentations for for Session 8B:


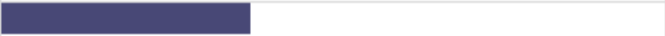

1 = Excellent, 2 = Good, 3 = Fair, 4 = Poor

Answer	1	2	3	4	Number of Response(s)	Rating Score*
Claudia Robertson					106	1.5
Geoff Manley					104	1.4
Paul Vespa					102	1.5

*The Rating Score is the weighted average calculated by dividing the sum of all weighted ratings by the number of total responses.

Please rate the following speaker presentations for for Session 9:



1 = Excellent, 2 = Good, 3 = Fair, 4 = Poor

Answer	1	2	3	4	Number of Response(s)	Rating Score*
Mark Lovell					116	1.6
Douglas H.Smith					126	1.5
Charles Hoge					121	1.6

*The Rating Score is the weighted average calculated by dividing the sum of all weighted ratings by the number of total responses.

Please rate the following speaker presentations for for Session 10:

1 = Excellent, 2 = Good, 3 = Fair, 4 = Poor

Answer	1	2	3	4	Number of Response(s)	Rating Score*
Geoffrey Manley					125	1.4
Ramona Hicks					121	1.5




*The Rating Score is the weighted average calculated by dividing the sum of all weighted ratings by the number of total responses.

EDUCATIONAL GOALS

The goals of the 2008 Symposium are:

1. To provide a forum for the presentation, discussion and feedback regarding the most recent findings in neurotrauma research and to encourage the interaction between both those new in the field and those with extensive experience in this area.
2. To present and discuss the findings of established neuroscientists engaged in other areas of research in order to encourage the participation in neurotrauma research of these experts as well as to provide the neurotrauma community the opportunity to incorporate these new ideas and concepts into their research.
3. To present and discuss the state of the art clinical management of both brain and spine/ spinal cord injury to clinicians and neuroscientists in order to encourage collaboration, communication, and participation in neurotrauma research and translate those concepts into clinical trials and clinical practice to improve outcomes in patients with neural injuries.
4. To provide hands on sessions for clinicians in the areas of brain and spine/ spinal cord injury to encourage the incorporation of the state of the art clinical techniques in reconstruction and multimodality monitoring in the care of the injured patient.
5. Describe the state of the art interventions, therapeutic strategies, and multimodality monitoring can be used to improve outcome after CNS trauma.
6. Describe how acute surgical intervention and later reconstruction can be utilized to improve the outcome of patients with traumatic brain and spine/ spinal cord injury.

IN YOUR OPINION, DID THIS PROGRAM SUCCEED IN REACHING THESE GOALS?

Answer	0%	100%	Number of Response(s)	Response Ratio
Yes			186	78.4 %
No			6	2.5 %
No Response(s)			45	18.9 %
Totals			237	100%

Please rank the most important factors in determining your attendance at NNS 2008:

1 = Least Important

Answer	1	2	3	4	5	Number of Response(s)	Ranking Score*
Scientific Program						185	3.6
Venue/Location						177	2.8
Faculty Speakers						182	3.2
Time of Year						171	2.5
Other						79	2.5

*The Ranking Score is the weighted average calculated by dividing the sum of all weighted rankings by the number of total responses.

TextBlock:

NEEDS ASSESSMENT SURVEY FOR FUTURE PROGRAMS

Please list two major areas of **KNOWLEDGE** or **SKILLS** that you feel must be addressed in the next few years:

100 Response(s)

Please let us know if you have any suggestions for **TOPICS** to be presented at next year's conference :

75 Response(s)

Please rank the deciding factors in attending NNS 2008:

(1=most important 4= least important)

1 = Most Important




Answer	1	2	3	4	5	Number of Response(s)	Ranking Score*
Topics						162	2.6
Location						155	3.0
Faculty						159	2.7
Time of Year						150	3.1
Other						68	2.9

*The Ranking Score is the weighted average calculated by dividing the sum of all weighted rankings by the number of total responses.

Which of the following educational components would you like to have MORE of in future programs?

Answer	0%	100%	Number of Response(s)	Response Ratio
General / Plenary Sessions			68	20.6 %
Breakout Sessions			52	15.8 %
Abstract Poster Sessions			60	18.2 %
Open Communications Sessions (Oral Abstract Presentations)			52	15.8 %
Hands-On Workshops			39	11.8 %
Networking Opportunities / Social Events			53	16.1 %
Other			5	1.5 %
Totals			329	100%

Which of the following future events do you plan to attend?

Answer	0%	100%	Number of Response(s)	Response Ratio
INTS / NNS 2009 Joint Symposium in Santa Barbara, CA (Sept 7-12, 2009)			135	64.2 %
SfN 2009			58	27.6 %
Other			17	8.0 %
Totals			210	100%

TextBlock:

LOGISTICS SURVEY

What HOTEL AMENITIES are most important to you?

Answer	0%	100%	Number of Response(s)	Response Ratio
Pool			48	8.6 %
On property variety of Dining Options			94	16.9 %
Fitness Center			62	11.2 %
Spa			13	2.3 %
Golf			4	<1 %
Tennis			2	<1 %
Children's Programs / Family-Friendly			22	3.9 %
Airport Shuttle			105	18.9 %
Internet Access			131	23.6 %
Proximity to Shopping, Tours, etc.			53	9.5 %
Other			19	3.4 %
Totals			553	100%

Please rate the OVERALL PROGRAM VALUE for the following:











1 = Excellent, 2 = Good, 3 = Fair, 4 = Poor

Answer	1	2	3	4	Number of Response(s)	Rating Score*
Meeting Dates / Time of year					177	1.8
Educational Content					179	1.4
Open Communications Sessions					173	1.7
Abstract Poster Presentations					175	1.7
Welcome Reception					148	1.7
WINTR Reception					94	2.0
WINTR Lunch Session					94	1.9
Student/Newcomer Networking Happy Hour					78	2.1
Networking Opportunities					148	1.9
NNS Business Meeting					67	1.9

*The Rating Score is the weighted average calculated by dividing the sum of all weighted ratings by the number of total responses.

Please rate the OVERALL EVENT LOGISTICS for the following:

1 = Excellent, 2 = Good, 3 = Fair, 4 = Poor

Answer	1	2	3	4	Number of Response(s)	Rating Score*
Website Information					175	1.5
Online Abstract Submissions process					148	1.5
Email Newsletters					156	1.5
Website Registration process					170	1.5
Organizer Communications					165	1.5
Onsite Registration & Staff					170	1.3
Hotel Selection					172	1.7
Hotel Service					171	1.8
City Selection					173	1.9
"All under one roof" Convenience					170	1.5

*The Rating Score is the weighted average calculated by dividing the sum of all weighted ratings by the number of total responses.

